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COARCTATION OF THE AORTA WITH PARTICULAR EMPHASIS UPON IMPROVED TECHNIQUES OF SURGICAL REPAIR

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DURING the 15-year period since successful surgical correction of coarctation of the aorta was first reported, extensive experience with this lesion has provided the basis for new concepts of surgical repair.[5, 10] Anatomic and physiologic variations of this congenital anomaly have received intensive study as a direct result of this surgical interest.[1, 2, 3, 9, 12, 13, 14, 16, 19, 22] Equally interesting in certain respects have been observations of acquired pathologic changes resulting from abnormal hemodynamics produced by the coarctation.[8, 17, 18, 20, 21] Ideal technical correction of this disorder must, on the one hand, take into account anatomic variations in location and extent of the process to establish normal aortic hemodynamics and, on the other hand, treat any acquired pathologic changes, such as local arteriosclerosis and aneurysm formation (Fig. 1). When surgical treatment was first introduced, limitations were placed upon selection of patients for operation based upon age, length of aortic occlusion, presence of associated pathologic lesions, such as atheromatous or aneurysmal disease, and other factors. In general, the primary

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objectives of therapy have been removal of the lesion and restoration of aortic continuity. As emphasized previously, however, certain modifications in this concept have evolved, influenced largely by the pathologic variations in the disease.[6] Since the major problem in this condition is concerned with hemodynamic disturbances produced by the occlusive process, the primary objective of therapy is correction of these disturbances with restoration of normal circulation. This may be achieved in most forms of the disease by extirpation of the coarcted segment with end-to-end anastomosis or with graft replacement (Fig. 2). In those associated with aneurysmal formation, extirpation of the lesion is essential. But in some of the more unusual types, particularly those associated with relatively long coarcted segments or with severe arteriosclerotic or calcific changes, the primary objective of therapy, that is, restoration of normal circulation, may be readily achieved by means of the bypass graft without removal of the lesion, thus avoiding some of the difficulties and hazards associated with excision and graft replacement (Fig. 3). In light of these observations this report is concerned with consideration of application of these concepts of surgical treatment and an analysis of our experience in a series of 171 cases of coarctation in which they were employed.

PROCEDURES USED

End-to-End Anastomosis.—In general, direct suture, with establishment of aortic continuity after resection of coarctation, has been employed most commonly for patients in the first two decades of life with short "adult" or postductile type stenosis (Fig. 4). The technique is now well standardized and does not require detailed general description. Following resection of the coarctation, further trimming or tailoring of the aortic ends should be performed to provide the widest possible lumen at the point of anastomosis. Most commonly, a double-ended suture of fine silk or Dacron is started in the lateral posterior segment of the anastomosis and continued posteriorly as an over-and-over suture placed from the inside or as a continuous everting mattress suture. The other needle is used to bring the continuous suture around laterally for completion of the anastomosis.

In young children it may be desirable to perform at least the anterior portion of the suture line in an interrupted manner to allow for future growth. Great care must be exercised in holding the occluding vascular clamps and placing the suture line because of the extreme fragility of the aorta and adjacent intercostal arteries. End-to-end anastomosis was used for aortic reconstruction in 89 per cent of children in the first decade of life and in 63 per cent of patients between the ages of 10 and 20 years. Beyond the age of 20 years, resection and direct anastomosis was employed in a minority of patients in each age decade of life (Fig. 5).

Wedge Resection and Repair.—Occasionally, particularly favorable anatomic factors in young individuals with very short isthmic coarctations may

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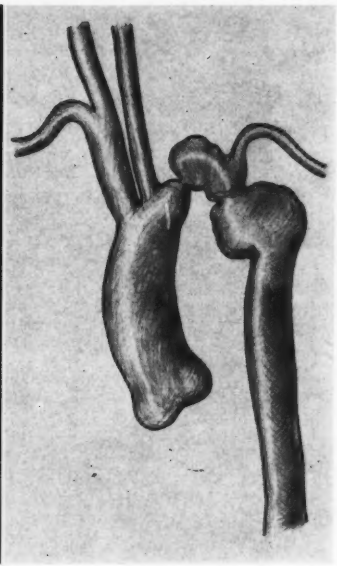

B.

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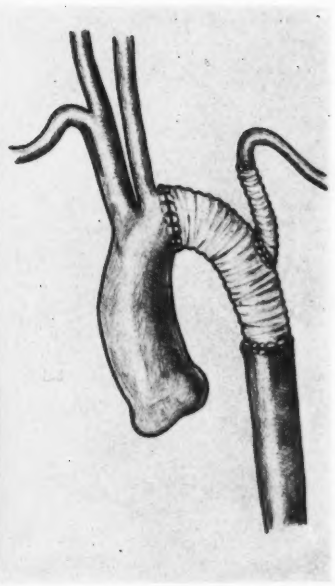


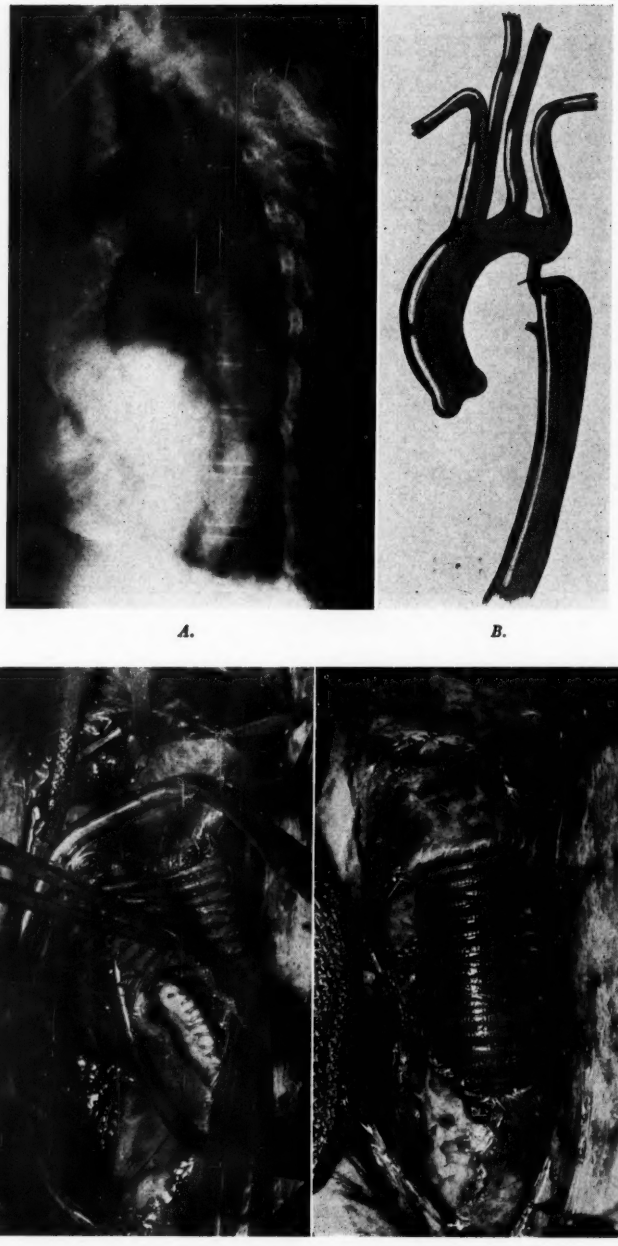
D.

Fig. 1.—*A*, Angioaortogram made before operation showing aneurysms associated with coarctation. Resected segment of aorta is superimposed.

B, Drawing which shows relation of aneurysms to coarctation and left subclavian artery.

C, Angioaortogram made after operation showing restoration of normal aortic lumen and contour.

D, Drawing which shows technique of aortic reconstruction.



A. *B.*

C. *D.*

Fig. 2.—*A*, Angioaortogram showing short isthmic coarctation in adult.

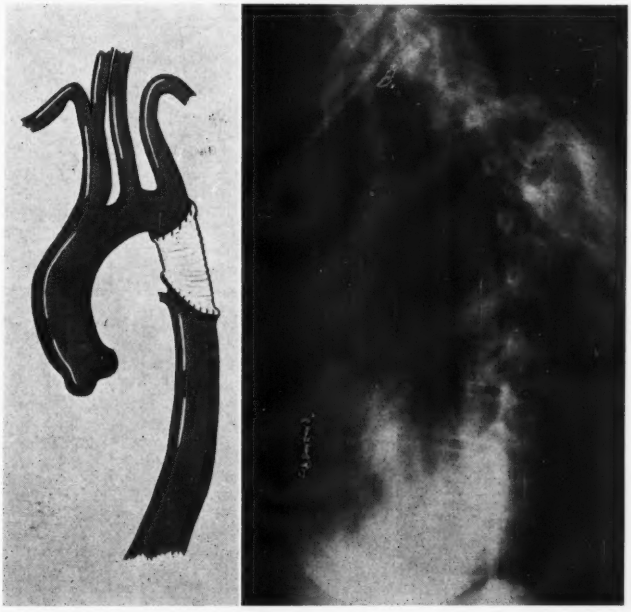
B, Drawing which shows location of coarctation.

C, Photograph made at operation showing placement of suture line for distal anastomosis between graft and aorta. Curved clamps have been applied in such a manner that wide anastomosis is possible after resection of coarctation.

D, Photograph made at operation showing functioning graft replacement of coarctation.

make wedge resection laterally with direct repair a satisfactory technical procedure. The suture repair is essentially similar to end-to-end anastomosis except that the posteromedial wall of the aorta in the stenotic area is not resected and forms one segment of the aortic wall opposite the suture line. Indications for application of this technique were uncommon in our experience as reflected by its use in only 3 per cent of 171 coarctations.

Resection and Graft Replacement.—End-to-end placement of graft to establish aortic continuity has had four general indications in this series: (1) long coarctations of the so-called infantile or preductile type, (2) coarctations



E. *F.*

Fig. 2.—Cont'd. *E,* Drawing which shows graft replacement of coarctation. *F,* Angioaortogram made following resection and graft replacement of coarctation showing normal aortic lumen and contour.

involving the aortic arch with stenosis extending proximal to the subclavian artery, (3) subisthmic coarctations involving the middle and lower third of the descending thoracic or upper abdominal aorta, and (4) any type of coarctation in which local degenerative changes made direct anastomosis hazardous. During the past 4 years, Dacron grafts have been used with extraordinary success. Graft repair minimizes necessary dissection of the distal aortic segment since a suitably curved vascular clamp may be employed to control this segment and exclude the origin of the intercostal vessels. This approach reduces hazardous dissection about fragile and diseased second and third intercostal arteries (Fig. 6). Similarly, a conveniently curved vascular clamp may be applied proximally in such a manner as to exclude varying

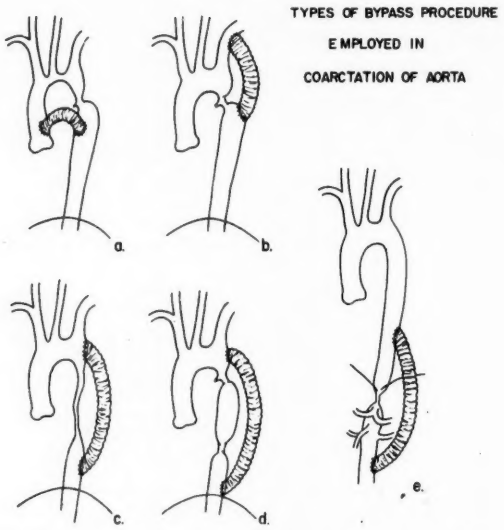
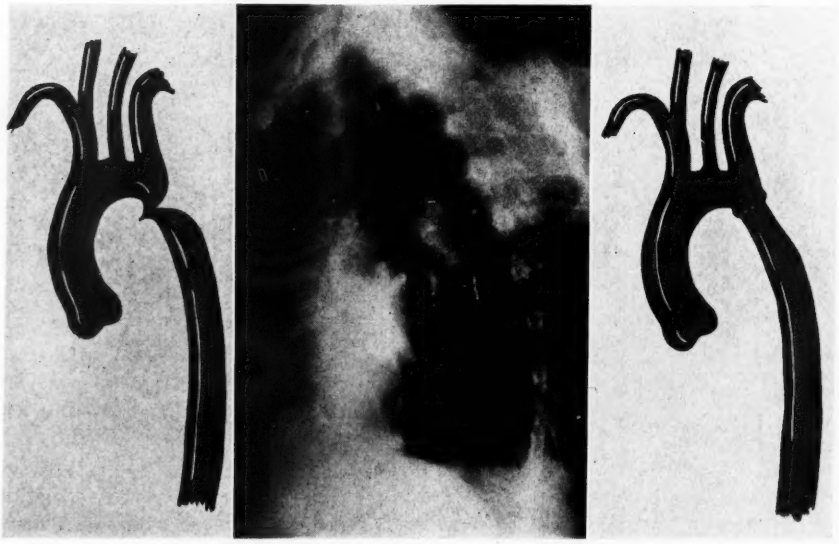


Fig. 3.—Drawings which show types of bypass procedure employed in coarctation of aorta.



A. *B.* *C.*

Fig. 4.—*A*, Drawing which shows short isthmic type coarctation in young patient suitable for resection and end-to-end anastomosis.

B, Angioaortogram made after resection and end-to-end anastomosis showing normal aortic lumen and contour.

C, Drawing which shows end-to-end anastomosis.

portions of the concave segment of the transverse aortic arch. Thus, by incising the aorta along its concave excluded wall for subsequent graft attachment, hypoplasia of the transverse aortic arch may satisfactorily be corrected. After selection and tailoring of a graft of suitable diameter, the proximal anastomosis is first completed using a continuous over-and-over suture of fine silk or Dacron. The graft is then cut to the appropriate length and obliquely tailored for distal anastomosis with the post-stenotic aortic segment. Just

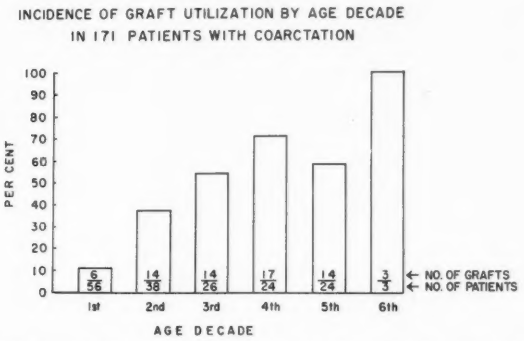


Fig. 5.—Graph which shows incidence of graft utilization by age decade.

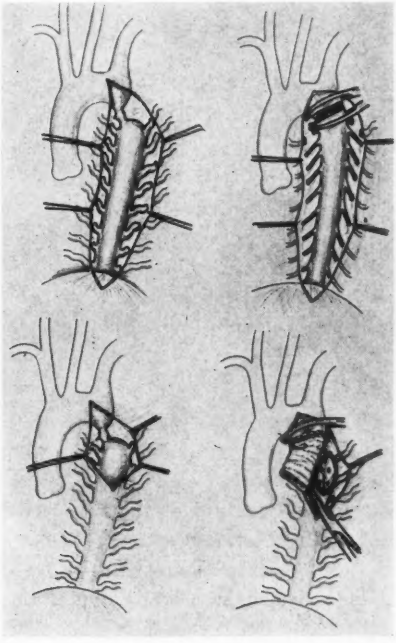


Fig. 6.—Drawings which compare extent of dissection usually necessary for resection and end-to-end anastomosis *(upper)* and resection with graft replacement *(lower)*.

before completion of the distal anastomosis, both proximal and distal occluding clamps should be momentarily released to flush the proximal and distal aortic segments. The distal clamp is removed first after completing the anastomosis. This allows inspection of the anastomosis under lower arterial pressure and provides a few moments to place additional interrupted sutures if indicated. The proximal aortic clamp is very gradually released to minimize bleeding and avoid sudden reduction in arterial pressure.

When the coarctation (or associated degenerative changes) involve the arch and the left subclavian artery must be isolated, it is usually desirable to establish aortic continuity first. The subclavian artery can be subsequently connected using a small exclusion clamp on the convex curve of the graft to facilitate anastomosis, either directly or by means of a side arm extension.

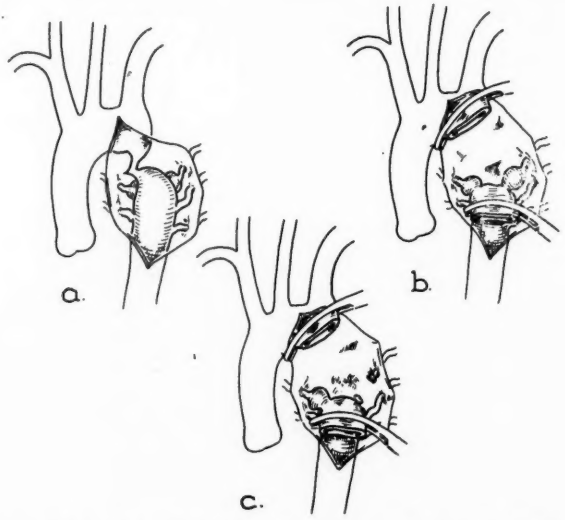
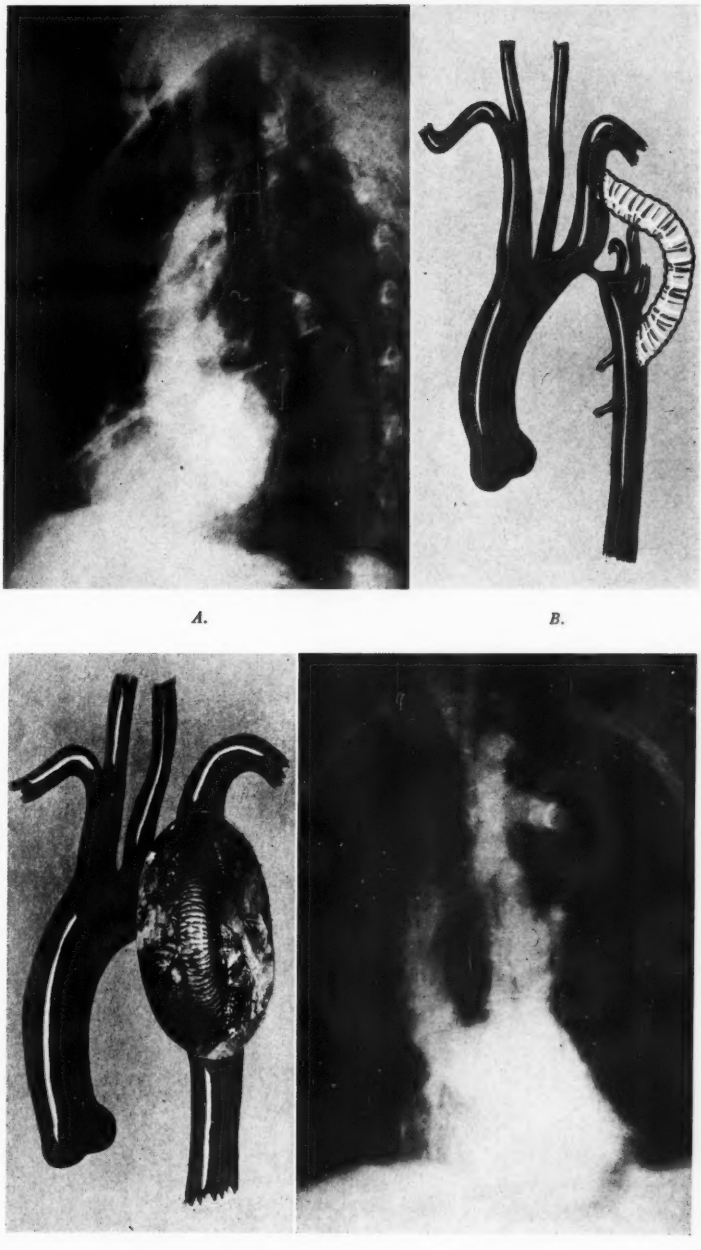


Fig. 7.—Drawings which show technique of exposure for resection of intercostal artery aneurysms. Coarctation initially exposed *(a)* and aorta cross-clamped and divided above coarctation *(b)*. With distal aortic segment elevated, intercostal aneurysms are easily exposed for resection *(c)*.

This approach reduces the period of aortic occlusion and provides attachment of the subclavian artery to the most desirable location on the graft, thus avoiding either tension or angulation.

Aneurysms of the upper intercostal arteries require resection for satisfactory surgical treatment. A technique which we have found of real merit in exposing and dissecting such intercostal arteries is application of occluding aortic clamps just above the coarctation followed by division of the aorta. Thus, with the distal aortic segment elevated, resection of aneurysmal intercostal arteries is greatly facilitated (Fig. 7).

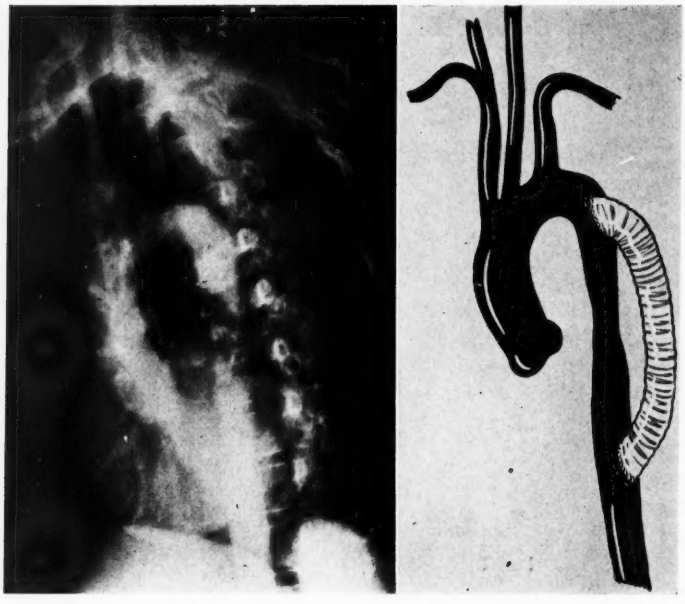
Aortic Bypass.—The bypass principle is useful in certain unusual circumstances (Figs. 8 and 9). There are a number of general indications in



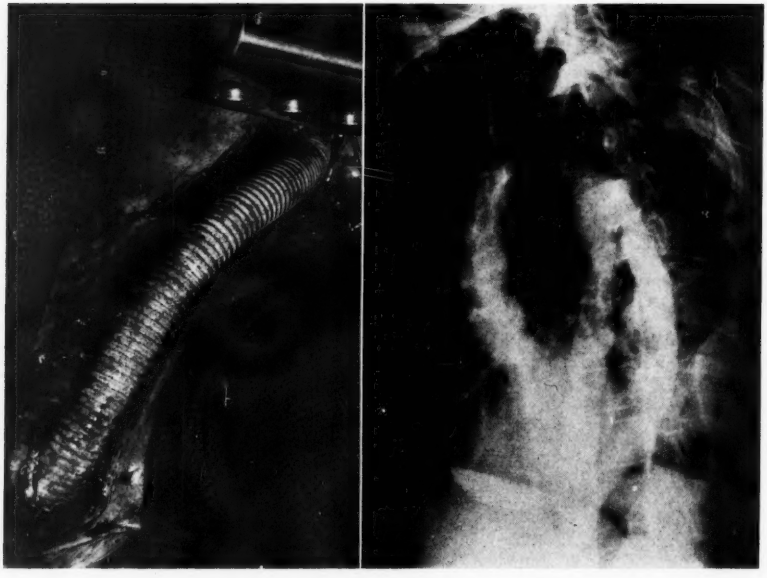
A. *B.*

C. *D.*

Fig. 8.—*A*, Angioaortogram showing short isthmic coarctation in adult with coronary artery disease.
B, Drawing which shows technique of bypass graft from left subclavian artery to aorta.
C, Drawing with superimposed operative photograph showing graft bypass.
D, Angioaortogram made after operation showing functioning bypass graft.



A. *B.*



C. *D.*

Fig. 9.—*A*, Angioaortogram showing long area of obliterative process involving descending thoracic aorta.

B, Drawing which shows location and extent of occlusive involvement and technique of graft bypass.

C, Photograph made at operation showing bypass graft.

D, Angioaortogram made after operation showing functioning bypass graft.

which application of this technique has appeared desirable: (1) situations in which resection of the coarctation may be hazardous, (2) poor-risk patients in whom this procedure facilitates expeditious surgical correction, (3) cases of long segmental involvement or multiple areas of stenosis, and (4) coarctations involving the lower thoracic and upper abdominal aorta. Appropriate exclusion clamps are applied to convenient segments of the aortic or subclavian wall proximally and the poststenotic aorta distally. With aortic blood flow essentially unaltered a linear incision can be made in the excluded segment for graft attachment. Proximal side-to-end anastomosis is completed first, followed by the distal end-to-side anastomosis to the aorta below the coarctation. Again flushing of the graft and gradual release of the proximal clamp is advantageous.

ANALYSIS OF CASES

During a period of 8 years, surgical correction of coarctation of the aorta was accomplished in 171 cases. The series consisted of 108 males (63 per cent) and 63 females (37 per cent). The age distribution was rather broad, extending from 19 children under the age of 1 year to 3 adults in the sixth decade. An analysis of age by decade has been compiled and reveals that almost half of these patients were over 20 years of age (Table I). Some of the clinical findings of particular interest are as follows. Hypertension in the brachiocephalic distribution was observed in 87 per cent of the cases. Neurologic alterations as a result of hypertension were seen in 25 per cent and ranged from relatively minor and reversible opthalmologic changes to hemiparesis. Cardiac decompensation had occurred in 36 per cent of the cases. Roentgenographic evidence of rib erosion or notching associated with enlarged intercostal arteries was seen in 61 per cent of the 171 cases. Accompanying patent ductus arteriosus was present in 44 cases (26 per cent).

TABLE I. ANALYSIS OF AGE IN 171 CASES OF SURGICALLY TREATED COARCTATION OF THE AORTA (NUMBER OF CASES IN EACH AGE DECADE)

	NUMBER	PER CENT
1st decade	56	33
2nd decade	38	22
3rd decade	26	15
4th decade	24	14
5th decade	24	14
6th decade	3	2
Totals	171	100

The anatomic location of the coarctation was in the region of the aortic isthmus in 152 cases (Table II). In 10 cases the stenosis began proximal to the left subclavian artery and involved the aortic arch. In 9 instances the coarctation presented itself in a subisthmic location, with 5 located in the middle and lower thirds of the descending thoracic aorta and 4 in the upper abdominal aorta. Not included in this series are an additional 15 cases with occlusive

processes produced by various etiologic factors with occlusion of the abdominal aorta between the renal and inferior mesenteric arteries.[15] Aortographic visualization to show level and extent of the coarctation seemed justified only in a minority of patients. This study is essential in patients with clinical features suggesting coarctation in unusual locations and in older adults to delineate associated degenerative changes such as aneurysm. Reduced blood pressure in the left arm suggests involvement of the transverse aortic arch. Classical findings that suggest subisthmic coarctation involving the lower thoracic or upper abdominal aorta are absence of murmur or, when present, located at a lower level; absence of notched ribs or, when present, notching of only the lowest ribs; absence of latissimus pulsation and an increased propensity for lower extremity claudication.

TABLE II. COARCTATION OF AORTA
(ANATOMIC LOCATION)

LOCATION	NUMBER	PER CENT
Isthmus	152	89
Arch	10	6
Subisthmus		
Descending thoracic aorta	5	3
Upper abdominal aorta	4	2
Totals	171	100

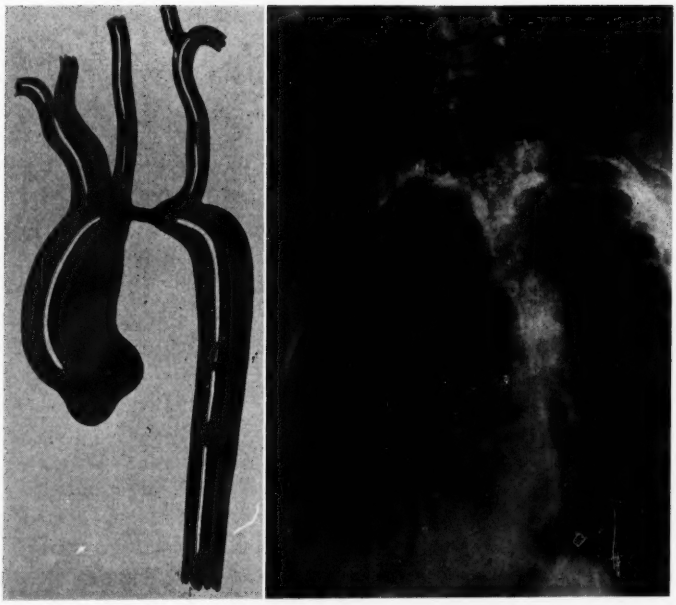
For purposes of analysis the type of surgical repair has been divided into four general categories. These divisions, together with the numbers in each, are as follows: end-to-end anastomosis 98 cases (58 per cent), wedge excision and suture 5 cases (3 per cent), resection and graft replacement 60 cases (35 per cent), and bypass with a graft 8 cases (4 per cent). The high incidence of graft application (68 cases) reflects the relatively large number of patients in the third, fourth, and fifth decades of life with attendant local degenerative changes in the aorta. In eight instances the origin of the left subclavian artery was resected which required suture of this vessel to the side of the graft. Bypass graft without resection was used in one coarctation involving the transverse aortic arch, in 2 poor-risk adults with isthmic coarctation, in two subisthmic coarctations, and in 3 patients with upper abdominal coarctation.

There were eight operative deaths in this series of 171 cases for a mortality rate of 4.6 per cent. Two children with coarctation of the upper abdominal aorta and associated atresia of the renal arteries died of sequelae arising from renal failure and 1 of these patients had endocardial fibroelastosis. Two infants died with pulmonary insufficiency associated with ventricular septal defects. Delayed hemorrhage from rupture or tear of the aorta near the suture line caused one death and coronary occlusion was fatal in one other case. Transfusion reaction and mesenteric arteritis accounted for two deaths, respectively. Follow-up studies revealed an additional four deaths 6 weeks to 1 year after operation. Two were the result of coronary occlusion, one was due to infection in the suture line of an end-to-end anastomosis, and one resulted from unknown causes.

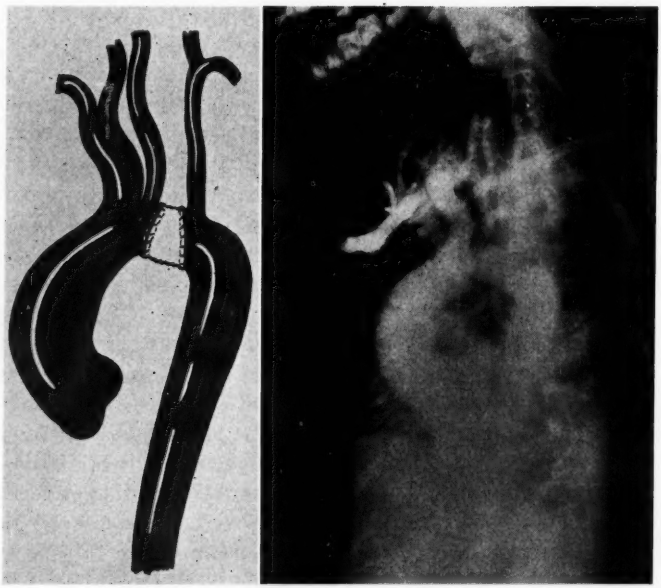
DISCUSSION

Experience in this series of 171 coarctations of the aorta appears unusual because it includes a high percentage of challenging surgical problems in patients who may be placed in the following groups—infants, patients over 30 years of age, and those with anatomic variants.[2, 11] There were 19 desperately ill infants under 1 year of age with intractable congestive heart failure and frequently associated cardiovascular malformations. Only two deaths occurred in this infant group for a mortality rate of 10 per cent and both deaths resulted from pulmonary insufficiency associated with large ventricular septal defects. Another group of patients presented unusual anatomic problems requiring a variety of far from standard surgical techniques for operative correction (Fig. 10). Two deaths in this group resulted from the anatomically and technically insoluble problem of diffuse bilateral renal artery hypoplasia. Finally, one of the most significant features in this series of surgically corrected coarctations was the high incidence of older adults. Forty-five per cent of patients were in the third to sixth decades of life. Two late deaths occurring in this group were due to coronary occlusion 6 weeks to 1 year after operation. Nonetheless the operative mortality of 4.6 per cent compares favorably with reports by others.[2, 11] Although elimination of these various challenging groups of coarctations could provide us with an extremely low mortality rate, the gratifying salvage of most such patients has continually reduced the number of patients in whom operative correction appeared contraindicated.

Our experience with the Dacron graft during the past 5 years has proved this material eminently satisfactory, not only for technical ease in handling during aortic reconstruction but, of more importance, for its long-term reliability.[7] It has maintained its tensile strength and has demonstrated no instances of late degenerative complications, such as aneurysm formation which is seen occasionally with homografts and nylon grafts. Application of graft reconstruction for aortic coarctation provides an almost limitless number of variations in surgical technique to meet individual problems. In most adults, reconstruction with a graft offers replacemment of the coarctation with an aorta of normal size and shape, thus reconstituting normal hemodynamics (Fig. 11). Probably the most significant factor in the high rate of graft application in our adult patients has been associated reduction in operative hazards with its employment. Dissection about the aorta may be greatly minimized and fragile intercostal arteries avoided. After applying suitably curved clamps to exclude the area of coarctation, the process can be removed together with diseased segments of the aortic wall and the aortic segments can be tailored obliquely to allow placement of a graft commensurate with aortic size. A secondary benefit derived from this approach is the technical facility with which the procedure can be accomplished with significantly reduced operative time. There appears now no justification for withholding use of a graft in adults if its employment will improve aortic reconstruction or provide a wider margin of safety. We are now so confident of the long-term results with



A. *B.*



C. *D.*

Fig. 10.—*A*, Drawing which shows unusual location of coarctation in transverse aortic arch.

B, Angioaortogram which shows coarctation between left carotid and subclavian arteries.

C, Drawing which shows technique of aortic resection and reconstruction with a graft.

D, Angioaortogram made after operation which shows reconstructed aortic arch.

the Dacron graft that we have little reluctance for its use even in children, when indication appears justified. During the past 3 years, incidence of graft utilization has remained at a high level exceeding 50 per cent application in each year (Fig. 12).

SUMMARY

Previous limitations placed upon selection of patients with coarctation for operation based upon age, length of the aortic occlusion, and presence of

A. *B.*

Fig. 11.—*A*, Photograph made at operation showing elongated isthmic coarctation. *B*, Photograph made at operation which shows graft replacement after resection of coarctation and adjacent hypoplastic aorta with reconstructed aorta of normal size and contour.

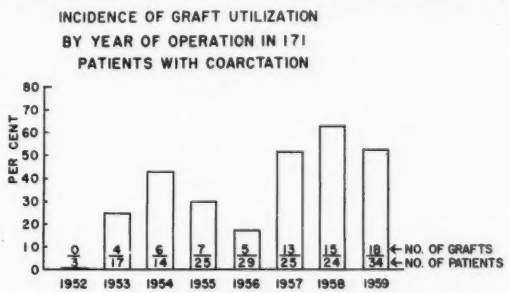


Fig. 12.—Graph which shows incidence of graft utilization by year of operation in 171 patients with coarctation.

associated pathologic lesions, such as atheromatous or aneurysmal disease, have been considerably altered by new concepts of surgical repair.

The primary objective of surgical treatment for coarctation is concerned with the hemodynamic disturbance produced by the occlusive process. This objective may be achieved in most forms of the disorder by resection of the coarcted segment with end-to-end anastomosis or with graft replacement. In those associated with aneurysmal formation, extirpation of the lesion is essential. In occasional instances the bypass graft may be the treatment of choice.

An analysis of 171 surgically treated patients with coarctation is presented in which newer surgical techniques have been applied to meet a number of challenging problems.

REFERENCES

1. Broden, B., and Karnell, J.: Coarctation of the Aorta; Aortographic Studies Before and After Operation, Acta radiol., Stockholm **50**: 1-61, 1958.
2. Burford, T. H., Ferguson, T. B., Goldring, D., and Behrer, M. R.: Coarctation of the Aorta in Infants: A Clinical and Experimental Study, J. THORACIC SURG. **39**: 47, 1960.
3. Clagett, O. T., Kirklin, J. W., and Edwards, J. E.: Anatomic Variations and Pathologic Changes in Coarctation of the Aorta; A Study of 124 Cases, Surg. Gynec. & Obst. **98**: 103, 1954.
4. Cooley, D. A., and De Bakey, M. E.: Resection of the Thoracic Aorta With Replacement by Homograft for Aneurysms and Constrictive Lesions, J. THORACIC SURG. **29**: 66, 1955; correction **29**: 216, 1955.
5. Crafoord, C., and Nylin, G.: Congenital Coarctation of Aorta and Its Surgical Treatment, J. THORACIC SURG. **14**: 347, 1945.
6. De Bakey, M. E.: Presidential Address: Changing Concepts in Thoracic Vascular Surgery, J. THORACIC SURG. **38**: 145, 1959.
7. De Bakey, M. E., Cooley, D. A., Crawford, E. S., and Morris, G. C., Jr.: Clinical Application of a New Flexible Knitted Dacron Arterial Substitute, Am. Surgeon **24**: 862, 1958.
8. Edwards, J. E., Christensen, N. A., Clagett, O. T., and McDonald, J. R.: Pathologic Considerations in Coarctation of Aorta, Proc. Staff Meet. Mayo Clin. **23**: 324, 1948.
9. Ellis, F. H., Jr., and Clagett, O. T.: Coarctation of the Aorta Proximal to the Left Subclavian Artery: Experience With Six Surgical Cases, Ann. Surg. **146**: 145, 1957.
10. Gross, R. E., and Hufnagel, C. A.: Coarctation of the Aorta; Experimental Studies Regarding Its Surgical Correction, New England J. Med. **233**: 287, 1945.
11. Groves, L. K., and Effler, D. B.: Problems in the Management of Coarctation of the Aorta, J. THORACIC SURG. **39**: 60, 1960.
12. Gupta, T. C., and Wiggers, C. J.: Basic Hemodynamic Changes Produced by Aortic Coarctation of Different Degrees, Circulation **3**: 17, 1951.
13. Hallenbeck, G. A., Wood, E. H., Burchell, H. B., and Clagett, O. T.: Coarctation of the Aorta; Relationship of Clinical Results to Cardiovascular Dynamics Studied Before, During, and After Surgical Treatment, Surg. Gynec. & Obst. **92**: 75, 1951.
14. Kirkendall, W. M., Culbertson, J. W., and Eckstein, J. W.: Renal Hemodynamics in Patients With Coarctation of the Aorta, J. Lab. & Clin. Med. **53**: 6, 1959.
15. Morris, G. C., Jr., De Bakey, M. E., Cooley, D. A., and Crawford, E. S.: Subisthmic Aortic Stenosis and Occlusive Disease, A. M. A. Arch. Surg. **80**: 87, 1960.
16. Nicks, R.: A Surgical Study of Coarctation of the Aorta, Brit. Heart J. **20**: 351, 1958.
17. Niedner, F. J.: Treatment of Aortic Isthmus Stenosis; Procedure in Patients With Advanced Changes of the Aortic Wall, Arch. klin. Chir. **287**: 438, 1957.
18. Oeconomos, N.: Dacron Prosthesis for Coarctation of Aorta With Aneurysm; Report of a Case in a Pregnant Woman, A. M. A. Arch. Surg. **77**: 181, 1958.
19. Scott, H. W., Jr., Collins, H. A., Langa, A. M., and Olsen, N. S.: Additional Observations Concerning the Physiology of the Hypertension Associated With Experimental Coarctation of the Aorta, Surgery **36**: 445, 1954.
20. Steinburg, I., and Sammons, B. P.: Aneurysmal Dilation of the Aortic Sinuses in Coarctation of the Aorta; Report of Two New Cases and Review of the Literature, Ann. Int. Med. **49**: 922, 1958.

21. Temesvári, A., and Árvay, A.: Some Clinical and Pathologic Aspects of Coarctation of the Aorta and Aortic Arch Syndrome, J. THORACIC SURG. 36: 247, 1958.
22. Wright, J. L., Burchell, H. B., Wood, E. H., Hines, E. A., Jr., and Clagett, O. T.: Hemodynamic and Clinical Appraisal of Coarctation Four to Seven Years After Resection and End-to-End Anastomosis of the Aorta, Circulation 14: 806, 1956.

DISCUSSION

DR. SAMUEL R. SCHUSTER, Boston, Mass.—This paper is especially interesting to us at the Children's Hospital in Boston, because we are just reviewing our own series of 505 coarctations which we have resected in the past 15 years.

In these cases we try to do a direct end-to-end suture. In 70 cases we inserted a graft. It is our belief that a graft should be inserted whenever necessary, but an end-to-end anastomosis is considered mandatory whenever possible.

We have attempted to do this by carrying out a more adequate mobilization of the aorta than was demonstrated in the paper today; a great deal of mobility can be gained by carrying the dissection of the aorta considerably farther down toward the diaphragm.

There has been no failure at the suture line, except in 2 patients with subacute bacterial endocarditis.

We have used homografts in all patients requiring grafting and have seen a fair amount of calcification in some of these homografts. However, we have not seen any aneurysmal distention of the homografts. We have not seen any occlusion of the homografts and, for the present, we see no reason to change to a prosthetic material.

We have performed one operation for coarctation of the abdominal aorta by using a homograft bypass, the bypass extending from above the diaphragm to below the renal vessels, with a good result.

DR. FRANCIS S. GERBASI, Detroit, Mich.—I rise to emphasize that coarctations of the aorta can occur elsewhere than in the thoracic aorta, and I think that we can make this diagnosis if we very carefully examine the entire chest and abdomen for bruits and pulsations.

I didn't do this 4 years ago, when I operated on a young girl 11 years old. I was sure I was going to find a coarctation in the upper thoracic aorta. I did a thoracotomy, and it wasn't there. I put my finger down along the aorta, and, sure enough, it was in the abdomen. We were using homografts at that time, and as I didn't have a graft long enough to do anything, I closed the chest.

A week later I did a retrograde aortogram [slide], which showed she had a coarctation of the abdominal aorta right in the area of the celiac axis. Here is the splenic artery [indicating], and there is the hepatic.

Two weeks later, through a thoracoadominal incision, I explored the entire abdominal and lower thoracic aorta. The stenotic area involved the aorta at the site where the celiac axis came off. I was very hesitant about doing an end-to-end anastomosis there because the celiac artery itself was very small, and I was sure it would thrombose if it were anastomosed to the side of the graft. I did what I thought was the next best thing—a bypass around the coarctation.

[Slide] It has now been 4 years since the operation. Before her operation she had a brachial pressure of 200/120 mm. Hg. It is now 110/70 mm. Hg, and she is asymptomatic.

DR. FRANK C. SPENCER, Baltimore, Md.—I rise to mention the use of the bypass principle in 2 unusual cases of long coarctation in the aortic arch. The atretic area began distal to the innominate artery, was 7 to 10 cm. in length, and involved the origins of the left carotid and subclavian arteries. Both patients were children operated upon at the Walter Reed Army Hospital; one was 2 and one was 12 years of age. In each child it was obvious on physical examination that the coarctation extended far proximally because the right carotid pulse was unusually prominent and the left carotid pulse absent; on angiography it could be seen that the coarctation began just distal to the origin of the innominate artery. The younger child also had some cyanosis of the lower extremities.

An anterior incision in the fourth intercostal space was used in order to have access to the ascending aorta. With the area exposed it was easily possible to insert a Dacron graft proximally into the aorta at the origin of the innominate artery and distally into the side of the descending aorta. A large ductus was also divided in the younger patient. Convalescence in both patients was uneventful.

DR. MORRIS (Closing).—I am indebted to the discussers for their interesting remarks.

In relation to Dr. Schuster's comments, I would certainly imagine that a preponderance of his cases were in children, and we certainly agree that end-to-end anastomosis is the treatment of choice. One objective in this presentation was to emphasize other techniques for challenging problems commonly seen in adults and in unusual anatomic types of coarctations.

With reference to abdominal coarctation, Dr. Gerbasi, we have had this problem also. We have had patients in our series with two separate areas of coarctation. In one, we initially missed the second coarctation and a second operation was necessary, but in the second case we were able to diagnose both lesions before operation and thus repair both concomitantly.

We also had some interesting cases such as Dr. Spencer described. In one particular case there was coarctation involving all of the transverse aortic arch, with extreme calcification of the aortic wall. We found two soft spots, one in the ascending aorta and one in the descending, and simply used a bypass graft directly across the arch.

FIVE-YEAR FOLLOW-UP STUDY OF CLOSED MITRAL VALVULOTOMY

J. Gordon Scannell, M.D., John F. Burke, M.D. (by invitation), Farrokh Saidi, M.D. (by invitation), and John D. Turner, M.D. (by invitation), Boston, Mass.

THE APPLICATION of new techniques to operations on the mitral valve makes it imperative to review the long-term results of closed mitral valvulotomy. It is particularly important to do this in order to provide a rational basis for open operation as an initial procedure in mitral stenosis.

One hundred consecutive patients who *survived* mitral valvulotomy at the Massachusetts General Hospital between June 1952 and January 1955 form the basis of this report. Seven patients who died of operation define the operative mortality during this period, but have been excluded from this follow-up study which is concerned with long-term results of operation. Five years after operation is taken as the end point of the study for each patient but information beyond that time is included whenever relevant. Information adequate to permit an intelligent evaluation at 5 years has been obtained for the entire series of 100 patients. Follow-up data in terms of an office or clinic examination by ourselves or a member of our own medical staff are available in 76. In 15, detailed clinical information has been supplied by the patient's physician in response to a personal request for information on the part of the authors, the remaining 9 patients have been examined in cardiac clinics of affiliated hospitals.

Since the series begins in June 1952, the first 17 patients undergoing mitral valvulotomy at this hospital have been excluded. This has been done in order to standardize the clinical material by eliminating the learning curve of the early cases so far as possible. It is interesting to note that of the first 17 patients, 3 have come to reoperation.

Without wishing to become involved in semantics, we use the term mitral valvulotomy as a convenient one to describe forcible dilatation by the index finger of a stenotic mitral valve with resulting split of the fused commissures. In the series here reported, if an adequate split of the commissures did not occur as a result of digital pressure, an attempt was made to cut the fused commissures with a knife or an Olivekrona pituitary scissors, while palpating

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the valve (15 of the entire series). At present, we prefer to use a mechanical dilator of the type advocated by Tubbs and Logan.[1]

In attempting to evaluate operative results, we have encountered the problem which troubles almost every group that reports the results of surgery for acquired valvular disease; namely, dissatisfaction with existing classifications and categories. We have, therefore, taken the usual course and set definitions which seem most applicable to the clinical material at hand.

In the preoperative evaluation we have recognized two groups of patients.

GROUP 1.—Individuals moderately to moderately severely incapacitated by their disease, of whom there were 74.

GROUP 2.—The severely incapacitated, of whom there were 26.

In general, these groups conform to American Heart Association classifications III and IV, respectively. The categories are intended to characterize the severity of disease, not the magnitude of operative risk which is an overworked and remarkably subjective term.

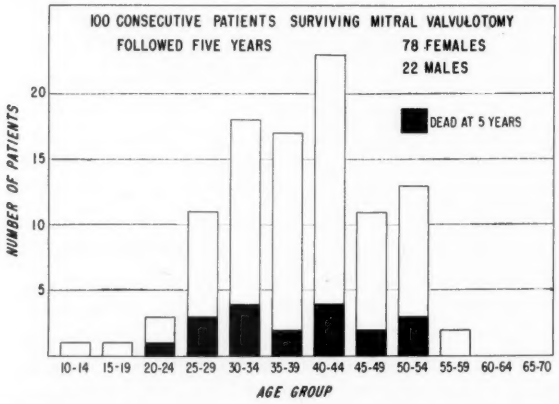

Fig. 1.—Age distribution of 100 patients surviving mitral valvulotomy, June 1952–January 1955.

In the postoperative evaluation of results, we have used the following scale of values.

1. The result is considered *excellent* if the patient is making no significant concessions to his disease, is free of symptoms under all reasonable conditions, tolerates periods of infection and stress (including pregnancy) well, and does not require salt restriction and cardiac medications. (An exception is the use of digitalis to control heart rate in the patient with atrial fibrillation.) Although such patients are generally under medical supervision, both the patient and the doctor are enthusiastic about the result.

2. The result is considered *good* if the patient is able to discharge his household or economic responsibilities without serious limitation, and requires only moderately strict medical supervision under conditions of physical stress or intercurrent infection. Both patient and physician are well satisfied with progress.

3. The result is adjudged *fair* if the patient is improved over his preoperative status, but still mildly or moderately incapacitated; requires careful medical supervision, restriction of salt intake, and occasional diuretics. Such patients usually are more enthusiastic about the result than the facts appear to justify.

4. The result is considered *poor* if the patient's condition is about the same as his preoperative status or worse.

5. Finally, *dead* is considered a category of result, irrespective of cause. In the present series, of 19 patients who died at or before 5 years, 11 died of congestive failure, 4 died of bacterial endocarditis, 1 each of cerebral embolus (at 4 mo.), pulmonary embolus (at 1 mo.), alcoholic cirrhosis, and sudden arrhythmia (patient up to that time [4 yr.] having done well).

The age distribution of the patients in this report is shown in Fig. 1. In general, it conforms to reported series elsewhere. Of greater importance, as will appear later under consideration of results, is the sex distribution. There were 78 females and 22 males. Calcification of the valve was found in only 15 per cent of the former, 55 per cent of the latter.

CLINICAL RESULTS

Of 100 consecutive patients who survived mitral valvulotomy, there were 81 alive 5 years after operation. (Five have since died.) Of these, 33 were rated as having excellent results and 28 good; to make a total of 61 unequivocally satisfactory results in terms of the initial operation. The results in 9 patients were considered fair and poor in 11 at 5 years. Of the latter group, 5 had undergone reoperation and survived, but in terms of the inital operation were considered to have poor results. Of the 19 patients who survived the initial operation, but who died within 5 years from causes enumerated above, 3 had undergone reoperation.

The 5-year results are presented graphically in Fig. 2. In addition, the results at yearly intervals are indicated. Thus, at 1 year, 78 patients were classified as good to excellent. At 2 years, 75 were so rated; at 3 years, 72; at 4 years, 67; and at 5 years, 61. The rather sharp drop of 23 per cent in the first year to 77 good to excellent results, with only a gradual decline of approximately 5 per cent per year thereafter, supports the clinical impression that the poor results generally become obvious within the first year—a phenomenon that is not surprising. After the first year there appears to be a steady attrition rate of about 5 per cent per year for the group as a whole. However, on an individual basis, if deterioration occurs, there is usually a rather obvious and sharp break in compensation. In our entire experience with reoperation for mitral stenosis, which now includes 8 closed and 18 open reoperations, the average interval between first and second operations was nearly 4 years.

It is extraordinarily difficult to find adequate controls against which to measure an operative series of patients with mitral stenosis. In Fig. 3 we have chosen to compare, in terms of survival, this group of 100 patients with

25 patients of so-called grade 3, and presumably comparable, severity, recently reported by Rowe, Bland, Sprague, and White.[2] By their definition, grade 3 means "moderate to moderately severe symptoms or one or more of the major complications (for example, embolism or endocarditis)." Since many of the patients of the present operative series were, in fact, under the care of the three senior authors of that follow-up study, and they had concurred in the indications for operation in the present series, it appears

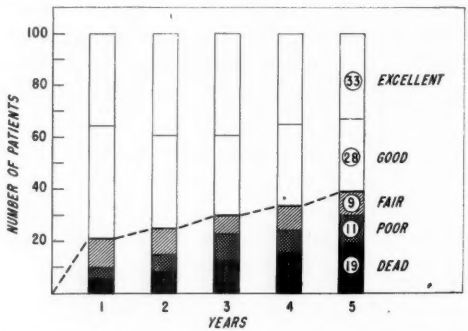

Fig. 2.—Clinical results (100 consecutive patients) at 5 years after operation, and at yearly intervals up to that time. See text for scale of values.

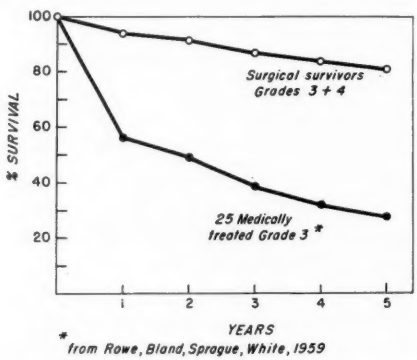

Fig. 3.—Comparison of survival rates in 100 consecutive patients with mitral stenosis treated surgically and medically.

reasonable to use their grade 3 patients as "controls." It is recognized, of course, that their patients were treated in the "pre-surgical era" and hence were not strictly a "control" group. Their 5-year survival rate for "grade 3" was 27 per cent as opposed to 81 per cent for the present series.

In Fig. 4 we have compared the 5-year results in patients severely incapacitated prior to operation with individuals moderately to moderately severely incapacitated. Of the 26 severely incapacitated, 50 per cent were rated good to excellent at 5 years, 29 per cent were fair to poor, and 31 per cent were dead. Of 74 patients less severely limited, 65 per cent were considered good to excellent, 20 per cent fair to poor, and 15 per cent were dead.

In Fig. 5 we have grouped results in terms of isolated factors at the time the patient came to operation in order to determine their bearing on long-term results. Thus, of 22 males, in only 36 per cent was the result adjudged good to excellent at 5 years, whereas of 78 females, 69 per cent were so rated. Of 24 patients noted to have significant calcification of the valve at operation (and this correlated only fairly well with pre-operative radiologic findings) only 29 per cent were doing well (that is, good to excellent) at 5 years, while

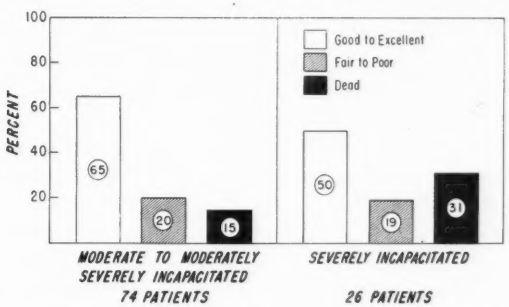

Fig. 4.—Clinical results at 5 years in 100 consecutive patients moderately and severely incapacitated at time of operation.

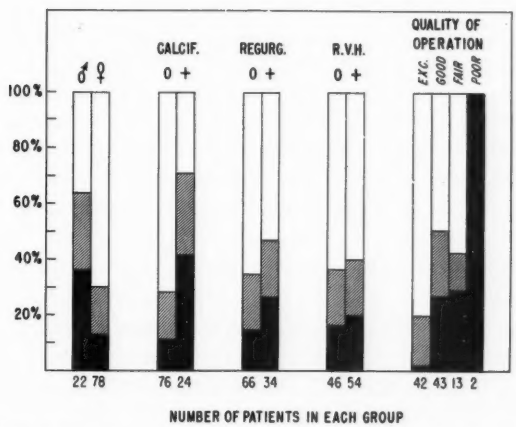

Fig. 5.—Clinical results at 5 years with reference to sex of patient, presence or absence of valvular calcification, postoperative regurgitation, right ventricular hypertrophy (ECG), and quality of operation. *White* = Good to excellent; *shaded* = poor to fair; *black* = dead.

70 per cent of the 76 patients without calcification were so considered. We do not mean to imply that calcification per se is the significant factor, but calcification associated with marked deformity and rigidity of the leaflets, and usually shortening of the subvalvular structures. It was surprising to find that the presence of regurgitation at the conclusion of operation had little effect on the outcome. In one or two instances this was true in spite of a considerable degree of postoperative regurgitation, but in the majority the insufficiency, though significant, was mild. Similarly the presence or

absence of right ventricular hypertrophy or right axis deviation in the preoperative electrocardiogram seemed to have relatively little determinant effect on the 5-year result.

Of greatest significance has been the operator's evaluation of the quality of operation; that is, the anatomic and functional result in terms of adequate size of orifice and mobility of leaflets, in addition to calcification and regurgitation. To some extent this evaluation is standardized, since the senior author participated in all operations of the entire series, but it must be acknowledged that what is accomplished by the operator in a closed mitral valvulotomy is a subjective evaluation on his part, corroborated at best by one or two assistants. When that evaluation was unequivocally excellent (42 cases), 78 per cent of the 5-year results were good to excellent. When there was some equivocation, but the quality of operation was considered good (43 cases), 49 per cent of the 5-year results were good to excellent. About the same end-results were achieved in the 13 patients in whom the operative result was considered "fair." It is in the latter two groups of cases that substantial improvement should be possible if mechanical dilatation is used to supplement digital maneuvers. The same may well be true for open operation if one assumes that removal of calcium and restoration of flexibility to the leaflets will have a lasting effect.

The problem of reoperation in patients who have undergone mitral valvulotomy is an important one. Of this series of 100 patients, 10 came to reoperation during the 5-year period of follow-up, or at some time thereafter. Rather than confine ourselves to these 10, we have thought that a more useful purpose would be served by reporting briefly our *entire* experience with reoperation for mitral stenosis. There have been 26 such operations carried out at the Massachusetts General Hospital. Eight of these operations were closed, 18 were open, using conventional methods of cardiopulmonary bypass and arrest.

The experience with closed reoperations may be briefly summarized as follows. Of a total of 8, 3 patients died of operation, 1 died 2 years postoperatively of congestive failure, 2 came to reoperation (open) at 14 and 16 months, respectively, and 2 are still doing well, 2 and 5 years after reoperation. Certainly this experience with closed reoperation was far from encouraging.

In the past 2 years we have carried out 18 open operations for recurrent mitral stenosis. The majority of these were in patients who had their initial operation at our own hospital. The average interval between initial and repeat operation was 3 years and 9 months. Six patients failed to survive. Five of these showed extreme degrees of pulmonary hypertension (the pulmonary arterial pressure equalled or exceeded the systemic) and extremely low cardiac output (Table I). Postoperatively all 5 were unable to maintain an adequate systemic pressure in spite of high peripheral venous pressure. This we have interpreted as a combination of right-sided failure and obliterative pulmonary vascular disease, making it impossible to maintain

an adequate cardiac output against a high pulmonary vascular resistance. All 5 succumbed within 24 hours of operation and at autopsy were found to have had excellent operative correction of their mitral stenosis.

TABLE I

PATIENT	AGE	SEX	INTERVAL BETWEEN OPERATIONS YR.	INTERVAL BETWEEN OPERATIONS MO.	CALCIFICATION AT SECOND OPERATION	CALCIFICATION AT FIRST OPERATION	GOOD TO EXCELLENT SPLIT FIRST OPERATION	CATHETER FINDINGS PAP	PAm	PC	CI	PAR	LIVING OR DEAD
Mu	42	F	5		+	+	+	80/35	45	25	2.4	11	L
Bu	30	F	8	8	+	Not known	Not known	80/47	63	20	0.9	48	L
Bl	39	F	2	5	+	0	0	70/34	43	32	2.2	5	L
Ga	50	F	3		+	+	0	80/40	54	38	4.4	4	L
De G	24	F	5		0	0	0	Study incomplete					L
Fi	35	F	4	10	0	0	+	90/50	65	28	2.0	17	L
No	49	M	2	11	+	+	+	65/32	43	24	2.0	10	L
Ga	28	F	1	6	0	0	+	84/38	61	31	2.8	11	L
Sw	33	M	2	11	+	+	+	—	40	15	2.0	13	L
Hy	35	F	4	2	0	Not known	Not known	40/18	25	17	2.0	4	L
St	41	M	5	1	+	+	0	No study available					L
He	43	F	7	11	0	0	0	120/50	70	27	1.5	29	L
Ha	34	F	1	4	+	+	0	110/50	65	27	2.0	19	D
Ro	19	F	5	8	0	0	0	115/58	80	25	1.7	32	D
Gr	27	F	3	9	0	0	+	Autopsy showed severe pulmonary vascular disease					D
Ho	42	F	1	7	Not known	+	Not known	120/48	62	—	—	—	D
Tu	31	F	2	5	+	0	0	59/28	39	20	2.2	9	D
Si	46	F	3	10	+	+	+	140/63	98	25	1.5	49	D

PAP = Pulmonary artery pressure (mm. Hg). PAm = Pulmonary artery, mean pressure (mm. Hg). PC = Pulmonary capillary pressure or left atrial pressure (mm. Hg). CI = Cardiac index (L./min./M.^{2}). PAR = Pulmonary arteriolar resistance—normal < 3.

Twelve patients have survived open reoperation and are presently doing well. Since the majority of these have been operated upon within the year no long-term follow-up is possible.

Of the entire series of 18 patients, calcification of the valve was pronounced in 10. Seven of these had had significant calcification of the valve at the time of their initial operation, which had been considered satisfactory in only 8 of the 18.

Returning to the original series (Table II), we find that 25 of the 100 patients had had peripheral emboli prior to operation. Twenty-one of the 25 showed atrial fibrillation at the time of operation. Of these 25, 6 had major emboli at the time of operation, 4 to the bifurcation of the aorta (all successfully removed) and 2 major cerebral emboli. Three patients of this group of 25 had late emboli; 1 at 3½ years, 1 at 20 months, and 1 had multiple visceral emboli to resume a pattern of peripheral embolization that she had exibited prior to operation. Three patients who had had no emboli prior to operation had operative emboli, 2 cerebral, 1 iliac. In addition, 2 patients without prior history of embolus had major cerebral emboli late; i.e., 4 months and 18 months after operation. That peripheral embolization is a major consideration in operative risk is indicated by the fact that of the 7 operative deaths which occurred during the period of this study, 4 were attributable to this cause.

TABLE II. EXPERIENCE WITH PERIPHERAL EMBOLI IN 100 CONSECUTIVE PATIENTS SURVIVING MITRAL VALVULOTOMY EVALUATED AT 5 YEARS

25 patients had peripheral emboli prior to operation
6 had emboli at time of operation
4 saddle emboli removed successfully
2 major cerebral emboli
1 fair recovery; 1 poor recovery
3 patients had late emboli postoperatively

SUMMARY AND CONCLUSIONS

The clinical result 5 years after operation has been evaluated in 100 consecutive patients surviving mitral valvulotomy. The result is considered unequivocally satisfactory, that is, good to excellent in 61; fair to poor in 20. This 5-year survival rate of 81 per cent represents a highly significant improvement over a comparable, though smaller, group of medically treated cases.

The long-term benefits of operation are most striking in patients moderately to moderately severely incapacitated by their disease, but there is a very rewarding salvage of patients severely incapacitated. The majority of poor results of operation become evident within the first year, but there is a steady attrition thereafter of recurrence of disability of about 5 per cent per year.

Three factors at the time of operation appear to have special prognostic significance: (1) the sex of the patient, the percentage of satisfactory results in women being greatly superior to that in men; (2) the presence of calcification and rigid deformity of the valves, indicating, in general, a poor prognosis; (3) the anatomic result of operation.

A review of the data suggests that the use of a mechanical dilator should improve the number of good results by increasing the number of anatomically excellent results, particularly in tough, fibrous valves. It is also a reasonable hypothesis that removal of calcium encrustations from a valve during the course of open operation may produce a greater number of satisfactory long-term results with greater initial safety than is possible at closed operation. Until the fate of decalcified valves is known, or the deposition of calcium can be controlled, this must remain supposition.

Of the present series, 10 patients have come to reoperation. The author's entire experience with 26 reoperations for mitral stenosis, 8 closed and 18 open, is briefly reviewed.

REFERENCES

1. Logan, A., and Turner, R.: Surgical Treatment of Mitral Stenosis With Particular Reference to the Transventricular Approach With a Mechanical Dilator, Lancet 1: 874, 1959.
2. Rowe, J. C., Bland, E. F., Sprague, H. B., and White, P. D.: The Course of Mitral Stenosis Without Surgery: Ten and Twenty-Year Perspectives, Ann. Int. Med. 52: 741, 1960.

(For Discussion, see page 799.)

THE TREATMENT OF ACQUIRED AORTIC STENOSIS BY VALVULOPLASTY

Donald G. Mulder, M.D. (by invitation), Albert A. Kattus, M.D. (by invitation), and William P. Longmire, Jr., M.D., Los Angeles, Calif.

THE PATIENT with acquired aortic stenosis remains a challenging surgical problem. Attempts to relieve the valvular obstruction by the currently recommended techniques have not been consistently successful. Our experience with the "blind" transventricular commissurotomy and the "open" direct vision commissurotomy has been disappointing. Unless these calcified, rigid valves can be mobilized, little functional benefit may be anticipated. Removal of the calcific deposits, originally suggested by Bailey,[1] will accomplish this objective. A technique has been developed for débriding the aortic cusps of the crucial, obstructing encrustations and for reconstructing the valve, when necessary, which we have referred to as aortic valvuloplasty.[2]

SELECTION OF CASES

Patients were selected for operation on the basis of clinical evaluation and the hemodynamic data obtained by left heart catheterization. Operation was not considered indicated unless the patient presented with symptoms of syncope, left ventricular failure, or angina. Progressive left ventricular enlargement on roentgenograms and electrocardiographic evidence of left ventricular hypertrophy were strong indications for operation. Calcification of the aortic valve and dilatation of the ascending aorta were confirmatory findings but were not considered essential.

Preoperative evaluation of potential surgical candidates by measurement of the pressure gradient across the aortic valve was considered to be essential to establish the diagnosis. The occasional patient with a classical "aortic stenosis murmur" caused by a diseased but nonobstructed aortic valve, who has angina due to coronary artery disease, will thus be spared a futile thoracotomy. Simultaneous cardiac output determinations have not been performed routinely. We still consider a gradient across the aortic valve of 50 mm. Hg or more as indicative of significant valvular obstruction. Under certain circumstances, such as a severely ill patient with a low cardiac output, operation has been advised despite a lower gradient. The presence of heavy aortic valvular calcification, associated mutlivalvular disease or acute cardiac decompensation, does not contraindicate surgical intervention, although the operative risk in these patients is greater.

However, active rheumatic fever or subacute bacterial endocarditis is definite contraindication to surgery.

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Read at the Fortieth Annual Meeting of The American Association for Thoracic Surgery at Miami Beach, Fla., May 11-13, 1960.

TABLE I. CLINICAL AND HEMODYNAMIC EVALUATION IN

	CLINICAL FINDINGS							X-RAY		
PATIENT	AGE AND SEX	HISTORY OF RHEUMATIC FEVER	HISTORY OF CHILDHOOD MURMUR	CHEST PAIN	SYNCOPE	DYSPNEA	HEART FAILURE	AORTIC VALVE CALCIFICATION	LEFT VENTRICULAR ENLARGEMENT	DIAGNOSIS
E. B.	43 M	0	0	0	++	++	0	++	0	A. S.
R. C.	49 M	+	0	++	++	+++	++	+++	++	A. S. A. I.
J. F.	42 M	+	0	0	+	++	++	++	++	A. S. (?AI) M. S.
R. F.	45 M	0	+	++	0	++	+	++	+	A. S.
B. G.	30 M	0	+	+	0	+	0	+++	+	A. S.
L. W.	48 M	0	0	0	+	++	+	+++	++	A. S. (?AI)
N. W.	39 M	0	0	++	0	++	0	+++	++	A. S.
D. Y.	17 M	?	0	+	0	+	0	0	0	A. S.
A. G.	35 M	0	+	0	?	++	0	+++	+++	A. S. A. I.
C. B.	46 M	+	+	+	0	++	+++	+++	+++	A. S. A. I.
H. M.	50 M	0	+	0	+	+	0	++	+++	A. S. A. I.
C. N.	23 M	?	0	+	0	++	0	0	++	A. S.
C. O.	55 M	0	0	++	++	++	0	++	++	A. S.
G. P.	48 M	+	+	++	+	+++	+	++	++	A. S.
W. V. N.	44 M	0	0	++	+	+++	+	+++	++	A. S.
B. J.	37 M	+	0	++	0	+++	0	+++	++	A. S.
M. W.	42 F	+	0	+	0	++	0	+++	++	A. S. A. I.
G. C.	46 M	0	0	++	++	+++	+	++	++	A. S.

Legend: A.S. = Aortic stenosis. M.S. = Mitral stenosis. A.I. = Aortic insufficiency.

PREOPERATIVE EVALUATION OF PATIENTS

Eighteen patients (Table I) represent our total experience to date in the treatment of acquired aortic stenosis by the operative procedure to be described. Five patients had significant to severe associated aortic insufficiency. There were 17 males and 1 female. The average age of the patients was 41.1 years. A murmur had been noted in childhood in 6 patients. A history of rheumatic fever was obtained in 6 patients, with a questionable history in 2 additional patients. These 2 patients, the youngest in the group, were the only ones who did not have gross, calcific valvular deposits. The disease did not appear to be of congenital origin because the valves were markedly thickened, rolled, and relatively rigid with fusion of the commissures. Furthermore, no murmur had been detected in childhood in these 2 cases. All patients gave a history of syncope, left ventricular failure, or chest

A GROUP OF PATIENTS WITH ACQUIRED AORTIC STENOSIS

PRESSURE MEASUREMENTS ACROSS AORTIC VALVE						RESULTS	
PREOPERATIVE		POSTOPERATIVE		GRADIENT (MM. Hg)			
LEFT VENTRICLE	ARTERIAL	LEFT VENTRICLE	ARTERIAL	PRE-OPERATIVE	POST-OPERATIVE		DURATION OF FOLLOW-UP (MONTHS)
174/0	100/56	112/4	112/56	74	0	Excellent	17
150/34	96/49	104/8	100/57	54	4	Excellent	16
180/30	120/60	103/8	100/62	60	3	Excellent	15
138/0	102/70	120/4	110/75	36	10	Excellent	12
200/10	110/70	148/4	140/88	90	8	Excellent	10
250/25	150/60	148/6	148/70	100	0	Excellent	9
225/10	110/60	148/8	120/72	115	28	Excellent	9
180/0	130/70	120/0	120/80	50	0	Excellent	12
240/10	108/50	92/0	92/64	132	0	Excellent initially; died 7 months postoperative with acute bacterial endocarditis	7
194/12	110/48	130/4	130/66	84	0	Excellent	8
266/30	85/38	130/4	110/52	181	20	Excellent	8
210/0	115/70	120/0	112/60	95	8	Excellent	6
200/0	110/60	—	—	(90)	—	Died at operation; irreversible fibrillation; calcium embolus to left coronary	–
180/0	130/77	90/4	90/70	50	0	Excellent	4
253/0	104/64	100/0	100/68	149	0	Died 3 days postoperative from severe central nervous system injury	–
186/0	116/60	110/4	104/65	70	6	Died suddenly 1 hour postop.; acute occlusion left coronary by embolic calcium fragment	–
220/10	120/52	140/10	140/85	100	0	Excellent	2
210/0	106/64	100/0	100/52	104	0	Died 2 weeks postoperative from severe central nervous system injury	–
			Average	90 mm. Hg	5 mm. Hg		

pain. Two patients had a questionable history of myocardial infarction. Roentgenographic examination revealed calcification of the aortic valve in 16 patients, and left ventricular enlargement was demonstrated in sixteen instances. Electrocardiographic evidence of left ventricular hypertrophy was present in every case.

Simultaneous pressure measurements in the left ventricle and femoral artery were made in all patients preoperatively (Fig. 1). The systolic gradient across the aortic valve ranged from 36 to 181 mm. Hg with an average of 90 mm. Hg. Simultaneous cardiac output studies were occasionally but not routinely performed.

OPERATIVE TECHNIQUE

The patient is placed supine and anesthetized with Pentothal sodium, nitrous oxide, and oxygen. The electroencephalogram and electrocardiogram are

continuously monitored. The systemic arterial pressure is recorded through a right radial artery cannula, and the superior vena cava pressure via a cardiac catheter passed up the right antecubital vein. Vasopressors are administered, if necessary, to maintain a satisfactory arterial pressure. Ventricular fibrillation is prone to occur in these patients if hypotension and lowered cardiac output occurs and is not promptly corrected.

A median sternotomy incision is preferred, which gives good exposure to the base of the heart and great vessels. The thymus is dissected off the pericardium and divided between its lobes up to the innominate vein. The pericardium is opened anteriorly to the right phrenic nerve, and the incision curved over

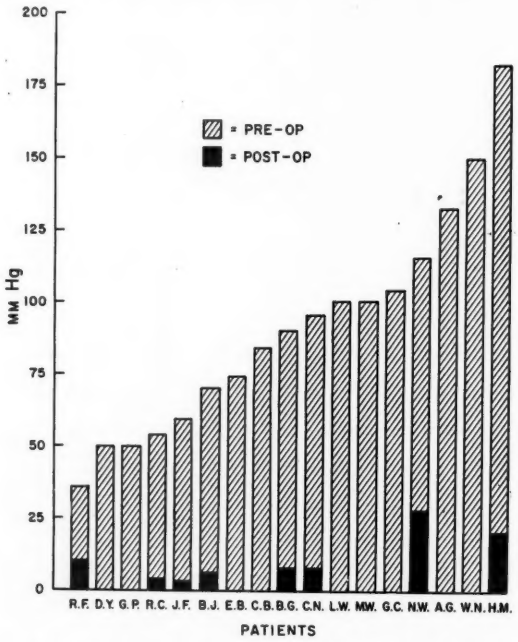

Fig. 1.—Bar graph which depicts the preoperative and postoperative systolic gradients across the aortic valve. One patient died at operation and no postoperative measurements were made.

the base of the great vessels toward the apex of the heart. Two purse-string sutures of 3-0 arterial silk are placed in the right auricular appendage. In the more critically ill patients, the right common femoral artery is exposed at this point through a longitudinal incision in the groin. After heparinization (1.7 mg. per kilogram), the patient is connected to the extracorporeal circuit (modified Kay-Cross disc oxygenator) by insertion of the femoral arterial cannula. The superior and inferior venae cavae and the aorta can now be safely dissected free and encircled with tapes, since cardiopulmonary bypass may be initiated without delay should the patient's condition deteriorate during

manipulation of the heart and great vessels. After a short stabilization run on full bypass, the ascending aorta is cross-clamped. The proximal ascending aorta is promptly opened through a longitudinal incision which is curved posteriorly (Fig. 2, *A*). The right coronary artery must be identified to spare it from injury. This hockey-stick incision gives satisfactory exposure (Fig. 2, *C*) and lessens the likehood of tearing the aortic incision by excessive retraction. The right and left coronary ostia are quickly identified and specially constructed perfusion cannulas are introduced (Fig. 2, *B*). In the initial 11 cases the heart was allowed to go into ischemic arrest. Currently, selective hypothermic coronary perfusion is used, although no instance of myocardial damage related to the former method was encountered. Approximately 1,200 to 1,500 c.c. of oxygenated, heparinized, hypothermic (15° C.) blood is perfused during a 5- to 7-minute period using an auxiliary De Bakey pump. Cooling is accomplished by passing the blood, which is diverted from the oxygenator, through a coiled polyethylene tubing submerged in ice-water. The blood is returned to the pump circuit by a right atrial cannula, or aspirated as it returns to the left ventricle. During the local cardiac cooling, the valvular deformity is carefully evaluated. The coronary perfusion cannulas are removed and attention is directed to repair of the valve.*

The critical obstructing and immobilizing encrustations are then removed by the following technique: An incision is made through the thickened endothelium along the base or annulus side of the calcific deposit (Fig. 3). An endarterectomy Freer elevator is used to establish the proper cleavage plane between the calcific mass and the underlying valve tissue. The débridement is made easier and more effective by starting at the base of the plaque deep in the cusp sinus. Although more difficult to visualize initially, it accomplishes two things: (1) the freed valve cusp does not tend to fold and lose its stability as the remainder of the encrustation is removed, and (2) no valve tissue is sacrificed by an incision into the leading edge of the calcareous mass, which may be partially covered by the rolled up cusp margin.

Care must be taken to prevent perforation of the diseased valve (Fig. 4, *A*, *B*, *C*). Spotty areas of through-and-through degeneration of the valve may not be apparent when viewed from the aortic side, but can be recognized by a dimpled area on the ventricular aspect of the valve; this must be débrided cautiously. Calcifications occur most commonly on the aortic side of the valve but may occur on the ventricular side as well. Occasionally, the coronary ostia are encroached upon by plaques; these should also be removed. Fibrous fusion at the commissures is incised by knife, either completely to the aortic wall or nearly so, depending upon the adequacy of the valve tissue. A tricuspid or biscuspid valve is fashioned as indicated by the valvular deformity in the individual case. Removal of the calcific crags often results in "unrolling" of the cusps so that the frequently associated aortic insufficiency may also be corrected. In other instances, after mobilizing the valve, it will still be necessary to form a bicuspid valve to achieve competency (Fig. 5, *A*, *B*).

*Continuous perfusion of only the left coronary artery with normothermic blood has been used in recent cases and, at present, is the technique we prefer.

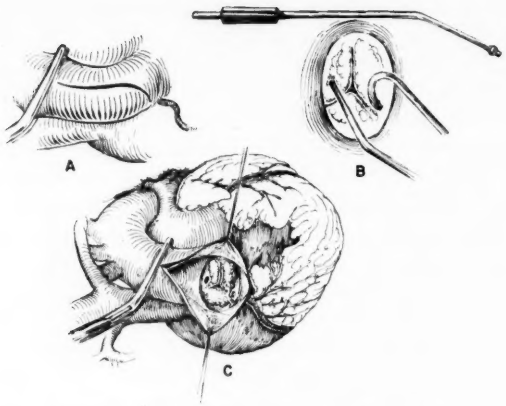

Fig. 2.—*A*, A longitudinal hockey-stick aortic incision is made.

B, Stainless steel coronary perfusion cannulas are inserted into the right and left coronary ostia, and the heart is perfused with oxygenated, hypothermic blood.

C, Good exposure is obtained with this incision which lessens the liklihood of tearing the edges of the aortic incision.

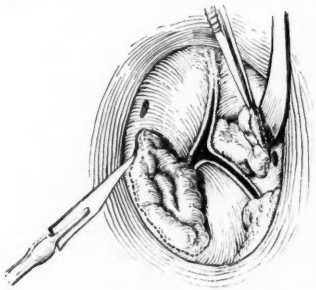

Fig. 3.—An incision is made at the base of the plaque. An endarterectomy Freer elevator is used to develop the proper plane between the calcareous mass and the underlying valve tissue.

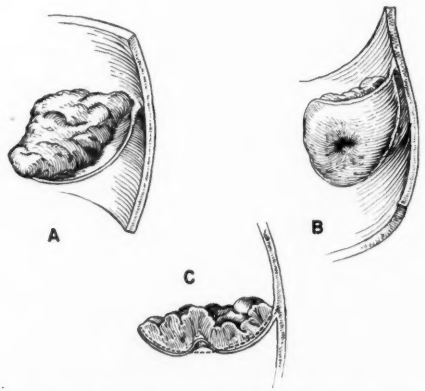

Fig. 4.—*A-C*. Three views of a diseased aortic valve cusp. No indication from aortic aspect *(A)* that dimpled area of through-and-through degenerative change is present which is apparent from ventricular aspect *(B)*. Cusp perforation *(C)* will result if radical removal of entire calcareous deposit is attempted.

Partial replacement or reinforcement by the insertion of a prosthetic cusp is occasionally indicated (Fig. 5, *C*, *D*). Decreasing the circumference of the aortic annulus by "taking a tuck" in the wall at one commissure has also been an effective method of correcting the associated aortic insufficiency (Fig. 6).

After the valvuloplasty has been completed, the left ventricle and root of the aorta are flushed with saline, the suture line is partially closed, and the remaining aortotomy is excluded with a Satinsky clamp. The aortic cross-clamp is then released and the patient is placed in parallel with the pump circuit by releasing the occluding vena caval snares. This prevents distention of the nonbeating heart by decompressing the coronary sinus blood into the caval catheters. The left ventricle and left atrium are carefully observed for distention which would indicate significant aortic insufficiency. If this occurs, the patient is promptly returned to full bypass and the repair revised, for

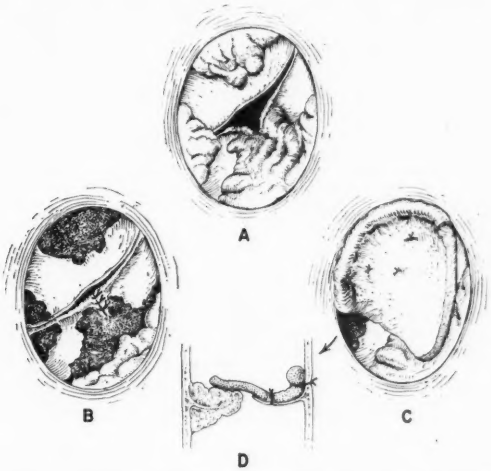

Fig. 5.—*A*, Typical deformed aortic valve with calcified encrustations which cause valvular stenosis and insufficiency.

B, After mobilizing the valve cusp, bicuspidization may achieve valvular competence as well as relieve obstruction.

C and *D*, When remaining valve tissue is inadequate despite débridement, prosthetic cusp is inserted.

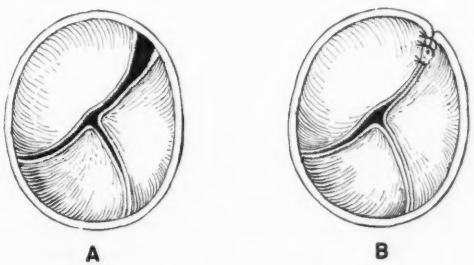

Fig. 6.—*A*, Aortic insufficiency may be due to defective valve tissue in one commissural region and/or annular dilatation.

B, "Annular tuck" technique will restore valvular competency.

it is virtually impossible to resuscitate the heart if significant aortic insufficiency is present. Following restoration of coronary blood flow, the heart action may return with a normal rhythm, but usually ventricular fibrillation is present which is reverted by electric shock.

As soon as the heartbeat is adequate, the extracorporeal circulation is discontinued, the aortotomy closure is completed, and all cannulas are removed. Protamine (2.0 mg. per kilogram) is given and the atrial and arterial incisions are repaired. The right pleural space is opened widely to accommodate any postoperative bleeding, and two chest catheters are inserted. The pericardium is left open. Prior to closure of the chest, repeat measurements of the left ventricular and systemic arterial pressures are performed.

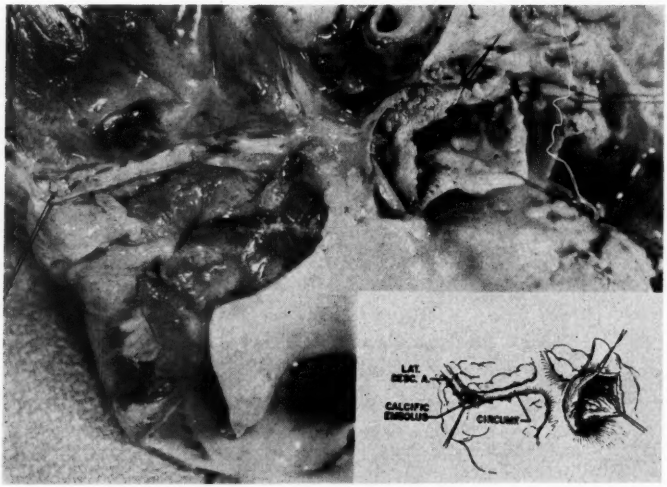

Fig. 7.—Finding at autopsy in patient (B. J.) who died suddenly 1 hour postoperatively. An embolic calcific fragment from the aortic valve completely occluded the left lateral descending coronary artery.

RESULTS OF OPERATION

There were four operative deaths among these 18 patients, a mortality rate of 22 per cent. One patient (C. O.) failed to survive the operative procedure. Removal of several large deposits of calcium had resulted in quite adequate aortic cusp mobility. As the operation was being completed, however, the heart action suddenly became very poor and ventricular fibrillation ensued which was irreversible. At autopsy, a fragment of calcium which had embolized from the valve was found to be completely occluding the left circumflex coronary artery. A similar cause of death was present in 1 other patient (B. J.), who died 1 hour postoperatively. Again the valvular reconstruction had been satisfactory. The patient was awake and maintaining a normal blood pressure when he suddenly developed irreversible ventricular fibrillation. In this instance the calcium embolus totally occluded the left lateral descending coronary artery (Fig. 7).

Two patients died from severe, progressive central nervous system damage, 3 days (W. V. N.) and 2 weeks (G. C.), postoperatively. The latter patient also had a massive upper gastrointestinal tract hemorrhage 6 days after operation and went into profound shock. Despite fresh blood replacement, the bleeding persisted and the patient underwent laparotomy. Suture ligation of a bleeding duodenal ulcer, pyloroplasty, and vagotomy were performed. Although he tolerated this episode surprisingly well, his condition steadily deteriorated neurologically, and he died 7 days following the second operation. At autopsy, no calcium emboli to the cerebral circulation could be demonstrated. However, large intravascular fat globules (Fig. 8) were noted in histologic sections of the brain. Whether these are related to the brain injury or not is speculative. An additional possibility is the ever-present hazard, especially in the treatment of lesions on the left side of the heart, of air embolism.

There was one late death (A. G.) which occurred 7 months after operation. This patient developed a bacterial endocarditis in the early postoperative period and was treated intensively for 6 weeks. At this time he was afebrile and blood cultures were negative. He returned to full activity and seemed to be doing very well until 1 week prior to his death when he developed a fulminating, recurrent bacterial endocarditis (Fig. 9).

Two patients (C. N. and G. P.) bled postoperatively from their aortotomy and re-exploration was necessary. Their subsequent convalescence was uncomplicated.

A bicuspid valve was initially present or created by suture obliteration of one commissure in 12 patients. A tricuspid valve was present or created by developing the obliterated commissure in 6 patients. Partial cusp replacement by a piece of compressed Ivalon was used in one instance (H. M.) to correct a moderately severe associated aortic insufficiency.

The 13 surviving patients have been followed from 2 to 17 months (average 10 months). The improvement in the physiologic classification of the cardiac status of these patients as determined by the criteria of the New York Heart Association is shown in Table II. Four patients were improved from Class IV to Class I, 2 patients were improved from Class IV to Class II, 5 patients were improved from Class III to Class I, and 2 patients were improved from Class II to Class I. There have been no syncopal episodes after operation, although 1 patient (H. M.), who had a history of a previous stroke, had a second stroke 2 months postoperatively. A transient hemiplegia was present for several days which cleared completely.

TABLE II. PHYSIOLOGIC IMPROVEMENT IN CARDIAC STATUS FOLLOWING VALVULOPLASTY

PREOPERATIVE	POSTOPERATIVE	NUMBER OF PATIENTS
Class IV	Class I	4
Class IV	Class II	2
Class III	Class I	5
Class II	Class I	2

A systolic murmur of decreased intensity remains in every case. Two patients have a residual diastolic murmur of aortic insufficiency.

At operation, prior to closure of the chest, repeat pressure measurements were obtained from the left ventricle and a systemic artery (see Table I). The postoperative systolic gradient across the aortic valve ranged from 0 to 28 mm. Hg with an average of 5 mm. Hg. In those patients in whom the gradient

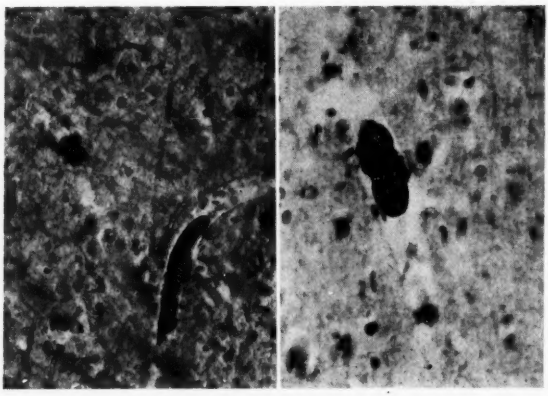

Fig. 8.—Histologic section of brain in patient (W. V. N.) who died 2 weeks postoperative from severe, progressive central nervous system damage. Large intravascular globules of fat are seen which were present throughout the brain.

Fig. 9.—Gross vegetations can be seen on aortic valve of patient (A. G.) who died 7 months after operation from fulminating, recurrent bacterial endocarditis.

was essentially abolished, the contour of the arterial pulse wave became relatively normal (Fig. 10). The classical anacrotic notch and slow "time to peak" of the arterial pulse tracing reverted postoperatively to a more normal contour.

DISCUSSION

There are those who continue to advocate the use of closed techniques in the surgical treatment of acquired aortic stenosis.[3, 4] A low operative mortality,

gratifying clinical improvement, and generally favorable hemodynamic changes have been reported.

Division of the commissures under direct vision using hypothermia as performed by Swan[5] and Lewis,[6] or using extracorporeal circulation as initiated by Lillehei,[7] have been accepted techniques.

The disadvantage of not being able to see and evaluate precisely what is being done to the valve during closed procedures would seem to be a significant one. Furthermore, it is not possible to correct the frequently associated aortic insufficiency; in fact, it may be made worse with this type of operation. Separation of the commissures under direct vision can be performed within the time limits of hypothermia. The addition of cardiopulmonary bypass would seem unnecessary if the objective of the procedure is only to cut or break open the commissures.

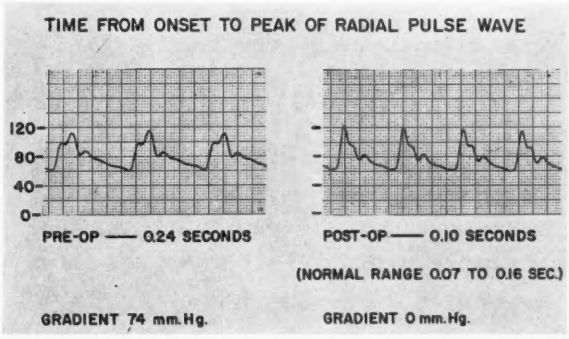

Fig. 10.—Changes in arterial pulse wave contour following valvuloplasty. Note the typical delayed "rise time" and anacrotic notch preoperatively compared to normal slope and contour of wave after operation.

We would agree with the concept of Bailey,[8] that only by removing the immobilizing calcific masses on the valve will cusp mobility be restored. In addition, added valve cusp length, sufficient to correct an associated valvular incompetence, may be gained as the plaques are removed and the underlying valve tissue is "unrolled." Unrelieved or surgically created aortic insufficiency is very poorly tolerated by the heart and may prevent its resuscitation. We have had no difficulty in re-establishing a normal beat in 11 patients who have had as long as 40 minutes of normothermic, ischemic arrest, providing a definite hemodynamic improvement was achieved. In no instance were there any late sequelae suggesting myocardial injury. In other patients (with pure aortic insufficiency) in whom the valvular incompetence was not satisfactorily corrected, restoration of heart action has been impossible following much shorter periods of arrest. Selective cardiac cooling has recently been adopted because of the reported myocardial protection it affords during prolonged cardiac arrest.*

*No longer used.

In this group of patients, it was always possible to remove much of the calcium from the cusp surface. The extent to which this can be accomplished will vary in each case, depending upon the severity of the degenerative valvular changes. A most gratifying restoration of function will be achieved in many instances where removal of two or three struts of valvular calcification restores the hinge action to the cusp. Occasionally, the valve will be almost replaced by a conglomerate mass of rock-like crags. Cautious, partial débridement of these amorphous encrustations often fails to restore sufficient cusp mobility to abolish completely the obstruction. Most commonly, the disease process falls between these two extremes, and the quality of the reconstructive procedure certainly is related in some degree to the severity of the valvular deformity. Regardless of certain limitations, it has been made possible, without exception, by painstaking removal of these plaques alone or in combination with additional valvular reconstruction (bicuspidization, annular tuck, prosthetic cusps), to improve distinctly aortic valve function.

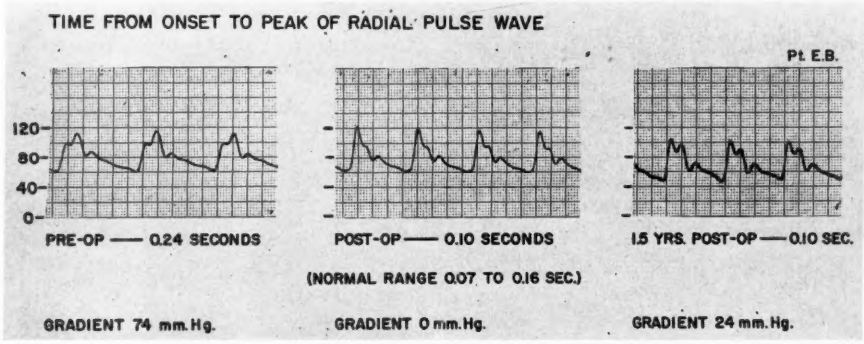

Fig. 11.—Repeat left heart catheterization in patient (E. B.), 1½ years postoperatively. Contour of arterial pulse wave remains normal. A gradient across the valve of 24 mm. Hg was recorded.

Total replacement would appear to be the ultimate ideal in the treatment of those patients with hopelessly destroyed aortic valves. Currently, such a clinically proved prosthesis is not available, but almost certainly will be in the near future. Even so, it is questionable whether the insertion of a foreign body at such a critical point in the cardiovascular system would be indicated in every case of aortic stenosis. For, if the patient's own valve can be restored to adequate function, this may be preferable to the insertion of a prosthesis.

The long-term results of this technique are still not known. There is no assurance that continued deposition of calcium and re-stenosis will not occur. Recatheterization of our first patient (E. B.), 1½ years postoperatively has been encouraging in this regard (Fig. 11). Preoperatively, he had a gradient across the aortic valve of 74 mm. Hg with a "time to peak" of the systemic arterial pulse wave of 0.24 second with the anacrotic notch typical of patients with aortic stenosis. Immediately postoperative, the gradient was completely abolished and the arterial pulse wave contour was normal with a "time

to peak'' of 0.10 second. One and one-half years later, he still has a normal pulse contour indicating no detectable delay in left ventricular emptying, although a gradient across his valve of 24 mm. Hg was demonstrated.

The future course and hemodynamic evaluation of these patients will be essential in order to assess the value of this procedure in the treatment of patients with acquired aortic stenosis.

SUMMARY

Eighteen patients with acquired aortic stenosis have been treated by aortic valvuloplasty during extracorporeal circulation.

The obstructing calcareous deposits were dissected from the valve tissue which resulted in relief of the obstruction and restoration of cusp mobility.

All patients presented with symptoms of syncope, left ventricular failure or angina, and demonstrated transvalvular pressure gradients averaging 90 mm. Hg.

There were four operative deaths: in two instances due to severe central nervous system injury, and in two instances to calcific emboli to the coronary arteries. One late death (7 months) was due to bacterial endocarditis.

The 13 survivors have all shown symptomatic improvement, and their average residual pressure gradient is 5 mm. Hg.

Hemodynamic measurement 1½ years postoperatively in 1 patient indicated that his operative improvement had been maintained.

REFERENCES

1. Bailey, C. P.: Discussion of Harken et al.[4]
2. Mulder, D. G., and Winfield, M. E.: Valvuloplasty for Acquired Aortic Stenosis, Ann. Surg. **151**: 203, 1960.
3. Glover, R. P., and Gadboys, H. L.: Seven Years Experience With Transventricular Aortic Commissurotomy, J. THORACIC SURG. **36**: 839, 1958.
4. Harken, D. E., Black, H., Taylor, W. J., Thrower, W. B., and Soroff, H. S.: The Surgical Correction of Calcific Aortic Stenosis in Adults, J. THORACIC SURG. **36**: 759, 1958.
5. Swan, H., and Kortz, A. B.: Direct Vision Trans-Aortic Approach to the Aortic Valve During Hypothermia: Experimental Observations and Report of Successful Clinical Case, Ann. Surg. **144**: 205, 1956.
6. Lewis, F. J., Shumway, N. E., Niazi, S. A., and Benjamin, R. B.: Aortic Valvulotomy Under Direct Vision During Hypothermia, J. THORACIC SURG. **32**: 481, 1956.
7. Lillehei, C. W., Gott, V. L., DeWall, R. A., and Varco, R. L.: The Surgical Treatment of Stenotic or Regurgitant Lesions of the Mitral and Aortic Valves by Direct Vision Utilizing a Pump-Oxygenator, J. THORACIC SURG. **35**: 154, 1958.
8. Bailey, C. P., and Zimmerman, J.: Advances in the Surgical Treatment of Aortic Stenosis and Incompetence *in* Advances in Cardiology, Basal/New York, 1959, S. Karger, vol. 2, p. 20.

(For Discussion, see page 799.)

PARTIAL AND COMPLETE PROSTHESES IN AORTIC INSUFFICIENCY

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DISEASES of the aortic valve present many anatomic and clinical syndromes. Aortic insufficiency with a dilated left ventricle in failure has been totally refractory to surgical intervention. Because of the clear diagnosis and grave prognosis of these patients, we are not only eager to intervene but have an obligation to undertake whatever we may regard as the most reasonable therapy. It is to this group that we address ourselves here.

Our efforts in treating aortic insufficiency have been in four categories: (a) circumclusion of the base of the aorta[12]; (b) direct plastic repair of the cusps; (c) Teflon* cusp extension; and (d) valve replacement with complete prosthesis.

CORRECTION WITHOUT PROSTHESES

Some years ago[12] we described a technique for *circumclusion* by placing a tapered suture that enlarged to a woven silk band around the dilated base of the aorta. This passed beneath the coronary ostia and, when drawn up, could reduce insufficiency by bringing cusps into better apposition. The anatomic and pathologic concepts of aortic insufficiency described in this connection have been useful.

We have not been successful with *direct plastic procedures* in patients with dilated ventricles in failure. Two patients have had this procedure. In each of these, the mobile noncoronary cusp was advanced into the central zone of insufficiency by suturing the right and left coronary cusps, respectively, to the commissure on either side of the noncoronary cusp. The commissure between the two coronary bearing cusps was then partially closed by sutures (Fig. 1).

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*C. R. Bard, Inc., Summit, N. J.

This reduced or eliminated regurgitation. Leaflet flexibility remained in the noncoronary cusp. The first patient could not be resuscitated after closing the aorta. The second patient had a more compact ventricle and did survive 36 hours but died suddenly. On the pulse duplicator at autopsy both had some degree of stenosis, although not of the size generally regarded as critical. According to Laplace's law, $T = P \times R$.* Therefore, if any gradient is produced across the valve it may cause fatal elevation in intramural tension in ventricles with large radii. This seemed to explain these failures. This concept must be borne in mind when "bicuspidalization" or other plastic procedures are considered.[13-15] Since this experience, we have used existing valve tissue only where the continuity of leaflets has been interrupted by subacute bacterial endocarditis or trauma.

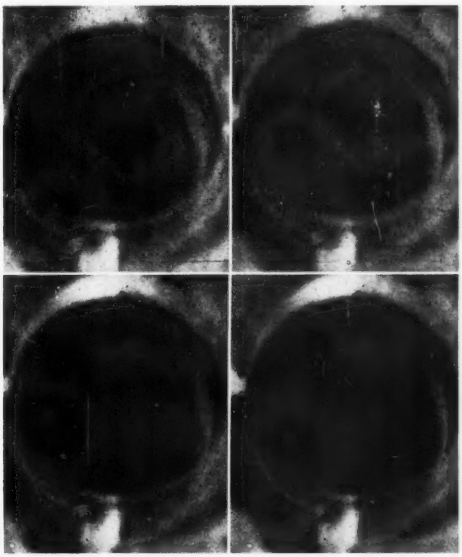

Fig. 1.—Pulsator films of aortic valve in autopsy specimen. A direct valvuloplasty was performed which corrected the aortic insufficiency but produced stenosis. The noncoronary bearing cusp is the cusp at the right.

The limitations of these plastic procedures led us to work on cusp substitution, then cusp extension, and eventually valve replacement.

Cusp substitution seemed feasible on the pulse duplicator and in the experimental animal. Teflon cloth seemed to be the most suitable material. It flexed well and could be solidly placed after excision of the neutral cusp. Others have had encouraging experience.[16, 17] However, Teflon cloth was abandoned for fear that it might become infiltrated with fibrous tissue and contract or roll into a stiff scar allowing regurgitation and stenosis. We even planted Teflon cloth in the abdominal wall of animals long enough to constitute a form of autograft. This, too, presumably has the disadvantages of late contraction and stiffening.

*Where T = intramural tension. P = pressure, and R = radius of the chamber.

CUSP EXTENSION

Nonwettable cusps of Teflon or polyurethane were unattractive in view of our experience with embolization from clot propagated at the junction of the endocardium with Lucite balls, bottles, and spindle baffles in mitral insufficiency.[18] Furthermore such materials require long fatigue tests. For example, if the opening and closure of a valve is one cycle, a heart rate of 100 beats a minute will undergo more than 50 million cycles a year. The testing equipment that we are using now cycles from 1,000 to 2,000 times a minute.

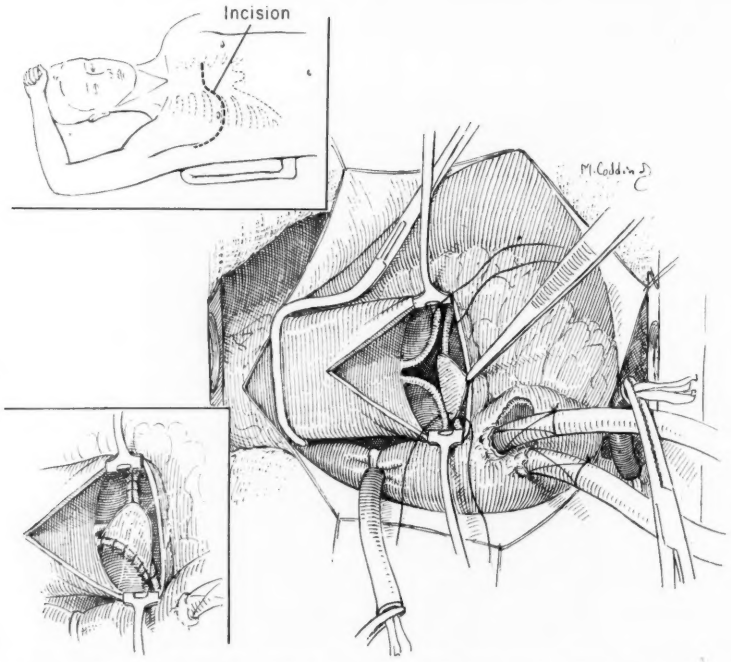

Fig. 2.—Extension of the neutral cusp. This technique was subsequently altered. A double thickness of Teflon patch was used to sandwich the edge of the leaflet so to prevent the sutures from cutting through.

Further acceleration seems impractical. Even this device has only recently been developed. Thus no flexion testing equipment has been available to give adequate information quickly.

Under these circumstances we have been eager to fatigue test material yet explore stiff cusp extension techniques that utilize the flexibility of the leaflets of the patient's heart and block reflux with a stiff woven Teflon patch extension (Fig. 2).

The noncoronary bearing or "neutral" cusp is not only the most mobile leaflet, but it has a characteristic position when the aortic valve becomes incompetent. It drops downward and backward. It may be incompetent by a loss of

substance centrally, by backward displacement, or by aortic base enlargement transversely. Also, as it falls to a lower level, incompetence is produced longitudinally (Figs. 3 and 4). This cusp is also in a right posterolateral position so that it and the corresponding sinus of Valsalva are accessible to surgical approach. Prosthetic extensions to this cusp therefore fall nicely below the coronary ostia. Finally, there is often fusion of the commissures between the coronary bearing cusps that serve as an excellent base against which the valve extension can seat.

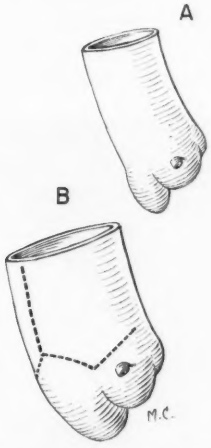

Fig. 3.—*A*, Profile of normal aorta indicating the lower position of the neutral cusp. *B*, Profile of aorta in aortic insufficiency indicating widening of the base and herniation of the neutral cusp.

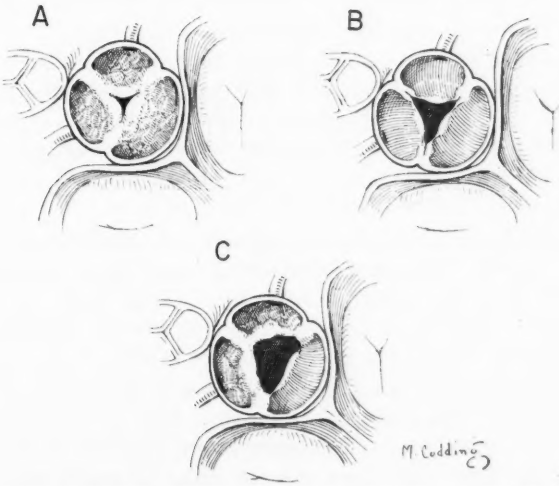

Fig. 4.—Common pathologic patterns of aortic insufficiency. *A*, Combined calcific aortic stenosis and insufficiency. *B*, Predominant aortic insufficiency, noncalcific. *C*, Predominant aortic insufficiency, calcific and noncalcific.

There have been four Teflon *cusp extension* operations. Of these 4 patients, 1 succumbed as the aorta was closed and the back pressure from the pump invaginated the extension. This was prevented in other patients by a pull-through traction suture placed at the apex of the Teflon that could be removed after closure of the aorta. Three other similar operations were performed and all patients survived. One of these patients died a year after his operation. He had done well for 10 months, then suddenly developed free aortic insufficiency and died soon thereafter in congestive failure. At autopsy the sutures were found to have cut through the cusp and released the Teflon extension. Subacute bacterial endocarditis in the interim may have contributed to the erosion and loss of the leaflet extension, but this loss was probably due to the fact that only a single layer of Teflon was sutured to the

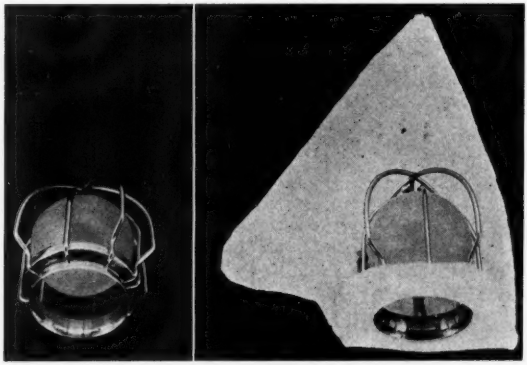

Fig. 5.—Caged-ball valve. Valve sutured in Ivalon.

free margin of the noncoronary bearing cusp (see Fig. 2). In subsequent operations the leaflet edge was sandwiched between two thicknesses of Teflon functionally similar to the original. Both of these patients are doing quite well 8 and 12 months after operation. Both are palliated and clinically improved. Neither have had complete correction of their regurgitation.

THE CAGED-BALL VALVE

As no Teflon leaflet extension was curative, we turned to the construction of a complete valve. In view of difficulties with fatigue studies, we sought materials that have been tested, namely, the Lucite balls of the Hufnagel valve,[19] or preferably the more recent, quiet, silicone rubber-covered ball. These have been in place in human beings since 1955.[20]

A stainless steel caged-ball valve has been built (Fig. 5).* The orifice is 1.98 sq. cm. The inner cage must allow the ball to move far enough away from the base so that it does not obstruct flow, but if it is too long the ball can move so far away that there may be delay in return and, thus, reflux. The inner cage is of close tolerance for rapid seating.

*Davol Company, Providence, R. I.

Studies of pulse contour indicate that the dimensions of this valve fulfill the hemodynamic requirements. With such narrow tolerance the ball extends beyond the inner cage and might touch the aorta. This would impair mobility and could cause stenosis or regurgitation. Therefore an outer cage was added to ensure freedom of ball motion. The ball is almost isobaric to blood (specific gravity—1.1499) so that it moves with minimal resistance in response to changes in the blood flow. For example, at a pressure of 80 mm. Hg acting downward on top of the ball, it will be displaced upward by a pressure change of 0.12 mm. Hg. Expressed in general terms, at this pressure level, opening requires only a 0.15 per cent pressure increment.

The valve is so constructed that it can be firmly anchored by Surgaloy* and covered by silk sutures in Ivalon† or Teflon backing. This, when cut into the shape indicated (Fig. 5), can be moved into position (Fig. 6) away from the ostia of the coronary arteries and yet provide an effective outflow tract. Further coronary clearance is gained by creating a small enlargement of the sinus of valsalva. The fundus of the noncoronary bearing cusp is excised (Figs. 6 and 7).

VALVE TESTING

Valves must not only be tested for fatigue, as discussed earlier, but for pulse contour, competence, resistance to flow, and trauma to formed blood elements.

A pulse duplicator has been constructed to study valve competence (Fig. 8). This mechanical device is composed of nine essential components. Part I is the actuator (Fig. 8, *6*) which contains a piston compressor that can vary its volume, rate, and duration of systole and diastole. Control is through the box (Fig. 8, *7*). The fluid is delivered into a rigid chamber corresponding to the ventricle (Fig. 8, *8*). From the rigid tube, a side-arm (Fig. 8, *9*) is provided where pressure contours can be recorded. The testing chamber (Fig. 8, *1*) contains the valve. Beyond the chamber housing for the valve is an elastic tubing that corresponds to the aorta (Fig. 8, *2*). Systemic pressure recording can be made at another side-arm (Fig. 8, *3*) which corresponds to the aortic arch. The character and degree of peripheral resistance can be altered by the position and degree of closure of a bar clamp (Fig. *8, 4*). Beyond the resistance clamp, the blood or circulating solution is returned to a reservoir (Fig. 8, *5*) in which there is a break in continuity so that the "arterial" system is interrupted to form a "venous" return.

This apparatus has been used in testing competence and pulse pressure contours for various types of valves. The caged-ball valve is competent when tested on this apparatus and produces pressure contours similar to those in the human being. These findings have been confirmed in our clinical experience (Fig. 9).

*Surgaloy—American Cyanamid Co., Danbury, Conn.
†Ivalon—Clay-Adams, Inc., New York 10, N. Y.

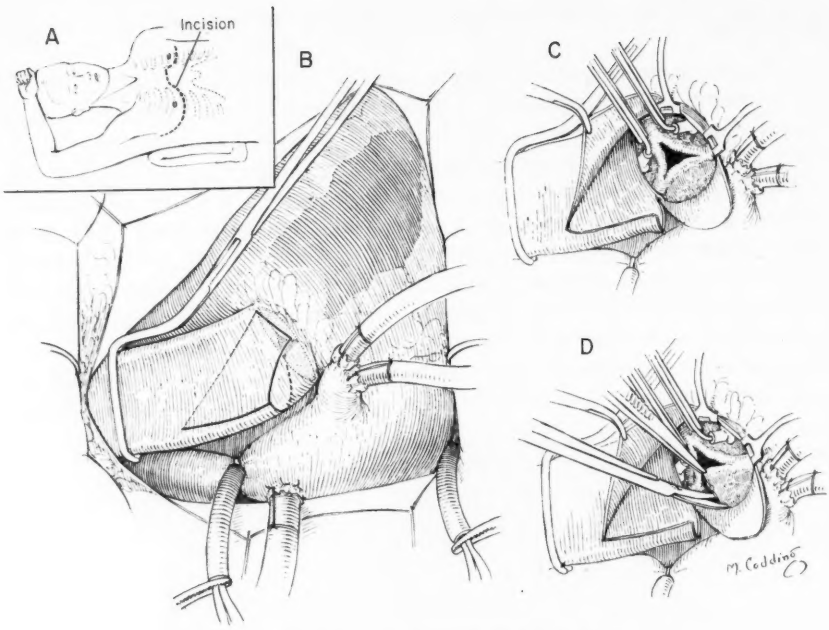

Fig. 6.—Technique of exposure of aortic valve.

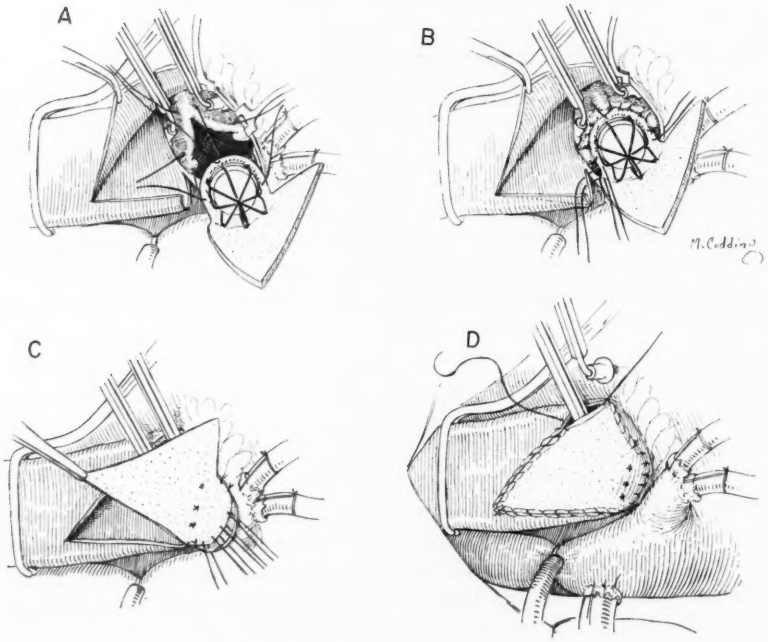

Fig. 7.—Technique of insertion of caged-ball valve.

Flow resistance through a valve is of prime importance. Continuous flow studies oversimplify the problem. However, at 10 L./min. the caged-ball valve offered no gradient. At 20 L./min., the gradient was 25 mm. Hg. Twenty liters a minute continuous flow would correspond to an average flow of 10 L./min. output at a pulse rate of 120, where time in systole equals diastole.

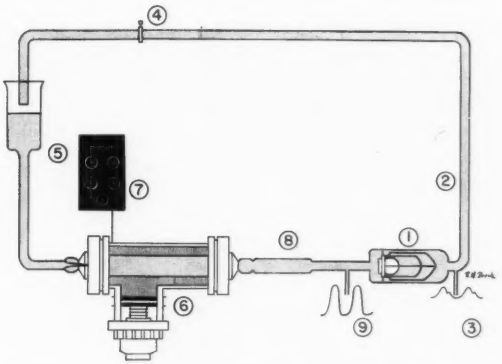

Fig. 8.—Schematic diagram of pulse duplicating system.

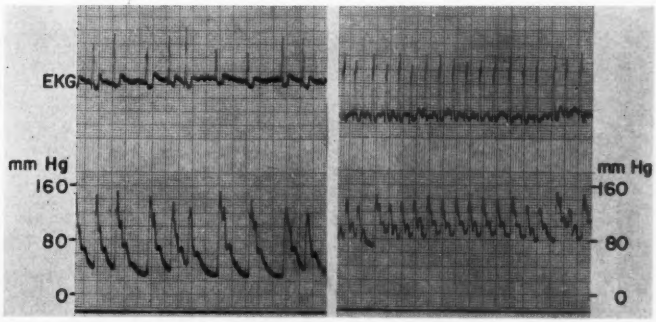

Fig. 9.—Pulse contour (Case 3) before and after surgery.

The caged-ball valve does not seem to be destructive to blood elements either experimentally or clinically.

Apparently the caged-ball valve is atraumatic, competent, enduring, quiet, offers little resistance to flow, and produces normal pulse contours.

CLINICAL USE OF THE CAGED-BALL VALVE

Our small clinical experience can be best presented in the form of case abstracts, omitting much significant clinical, pathologic, hemodynamic, hematologic, and metabolic data. After the case presentations a review of the dominant problems of the whole group clarifies the evolution of this technique.

A. *Case Reports.*—

CASE 1.—D. L. (PBBH No. 4P339), a 27-year-old man, had rheumatic fever at the age of 14. This was followed by evidence of severe aortic insufficiency accompanied by cardiomegaly, exercise limitation, and rare episodes of transient blindness. During the 2 years prior to hospitalization he had three episodes of congestive failure followed by increasing dyspnea, angina, paroxysmal nocturnal dyspnea, fatigue, and orthopnea. At the time of admission he was completely incapacitated in spite of intensive medical therapy.

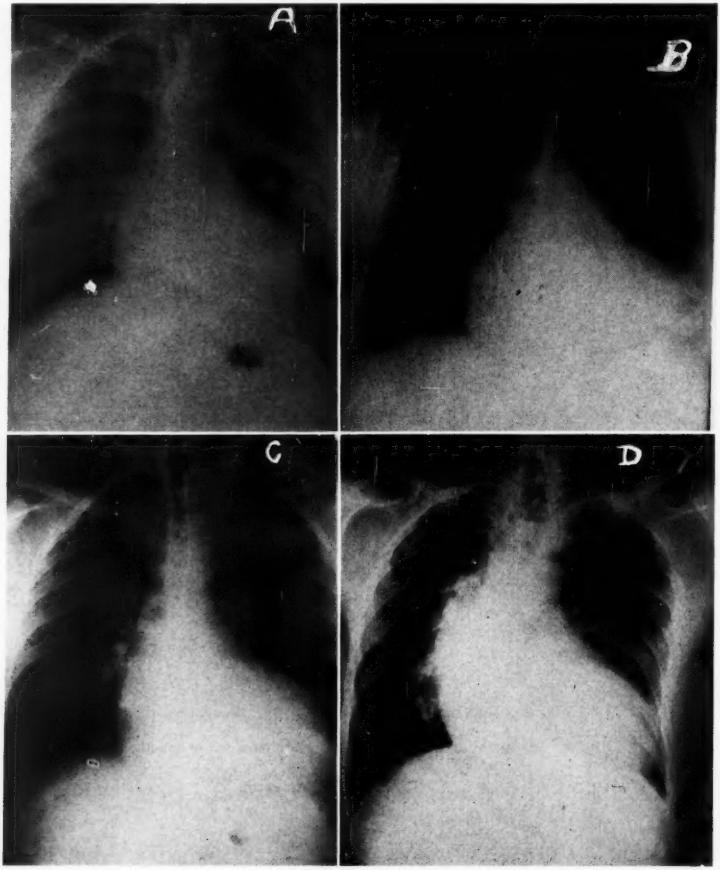

Fig. 10.—Posteroanterior roentgenograms of patients who died during insertion of prosthetic valve.

Physical examination: The blood pressure was 210/0 mm. Hg. The heart was enlarged to the anterior axillary line. There was a diffuse apical impulse and a left ventricular heave. There was a systolic ejection click and an accentuated pulmonary second sound. There were Grade 2 systolic and Grade 3 diastolic aortic murmurs. Grade 2 presystolic rumble was present at the apex. The liver was palpated two fingerbreadths below the right costal margin. There was no peripheral edema.

Roentgenograms (Fig. 10, *A*) and fluoroscopy revealed cardiomegaly with marked left ventricular enlargement. No calcification of the aortic valve was seen. The rocking beat of aortic insufficiency was no longer present. This was interpreted as an ominous sign.

Electrocardiogram showed normal sinus rhythm, left bundle branch block, and left ventricular hypertrophy.

Hospital course: He developed bouts of supraventricular tachycardia and angina decubitus persisted. A low-grade fever led to the diagnosis of and treatment for subacute bacterial endocarditis, although blood cultures were negative. He lost 3 kilograms in weight on a diuretic program.

At operation on bypass the patient was cooled to 26° C. with the Brown-Harrison heat exchanger. Ventricular fibrillation occurred at 28.4° C. and the aorta was cross-clamped and opened at once. Hypothermic arrest was brought about using intracardiac and extracardiac Ringer's solution at 4° C. The aorta was occluded for a total of 63 minutes while the caged-ball valve was inserted (see Figs. 6 and 7). With the resumption of coronary flow at release of the aortic clamp, transient defibrillation was accomplished but marked dilatation occurred followed by failure and again by fibrillation. In the dilated ventricle, defibrillation was impossible. The cannula had been placed in the left auricle and did not decompress the ventricle adequately.

Postmortem examination: The empty heart weighed 750 grams. There was right and left ventricular dilatation and hypertrophy and a partial occlusion of the anterior descending branch of the left coronary artery by an atheromatous plaque.

CASE 2.—M. R. (PBBH No. 4P565) is a 32-year-old woman without a history of rheumatic fever. A murmur was first heard at age 9 and since then she noted fatigue and slight exertional dyspnea. At 18 her only pregnancy was complicated by glomerulonephritis. At 26 she developed increasing fatigue, dyspnea, and transient left anterior chest pain. There were "weak spells" but no true syncope. Three years later, in the presence of progressive dyspnea, paroxysmal nocturnal dyspnea, and occasional ankle edema, she was admitted to another hospital for cardiac evaluation. Data from percutaneous dorsal catheterization of the left heart were incomplete because of syncope and shock. On the basis of clinical evaluation and available catheter data, transaortic valvuloplasty was performed. Stenosis of approximately 1.2 sq. cm. with regurgitation was partially corrected by finger fracture valvuloplasty. Transient improvement was followed by progressive limitation from dyspnea, orthopnea, and peripheral edema. At the time she was admitted for re-evaluation, she had dyspnea on less than one flight of stairs and angina decubitus.

Physical examination: The lungs were clear and the liver edge palpable at the costal margin. The blood pressure was 150/80 mm. Hg and the heart was in normal sinus rhythm. A left ventricular impulse was felt in the anterior axillary line. Grade 3 aortic systolic and diastolic and Grade 1 mitral diastolic murmurs were present. The aortic second sound was diminished.

Roentgenograms (Fig. 11, *A* and *B*) and fluoroscopy demonstrated moderate left ventricular enlargement and no calcification of the aortic valve. A definite rocking beat was present. The transverse cardiac diameter was within accepted normal limits.

The electrocardiogram showed normal sinus rhythm and left ventricular hypertrophy.

At transseptal cardiac catheterization, no gradients were noted across the aortic or mitral valves at a flow of 5.1 L./min. The dye dilution curves indicated regurgitation. The left ventricular end-diastolic pressure was 12 mm. Hg. Mild oxygen desaturation (89 per cent) was present in the sedated basal state.

Ventilatory studies and laboratory analyses, for serum electrolytes and blood constituents, were within normal limits.

Hospital course: Intensive medical therapy resulted in a weight loss from 69 to 66 kilograms. At *open operation*, on bypass, there was free relative aortic insufficiency from backward and downward displacement of the neutral cusp. The right coronary cusp was torn

through its lateral commissure to the base of the fundus. The leaflet complex could prolapse in diastole. Using a heat exchanger, body temperature was lowered. At 29° C. the heart went into ventricular fibrillation. During the ensuing 64 minutes of total bypass, at flow rates averaging 3,600 c.c./min., the patient's temperature remained at 28° C. The left ventricle was decompressed through a No. 38 plastic catheter. Adequate bilateral coronary perfusion was not attained. After the caged-ball valve had been placed (see Figs. 6 and 7) and as

Fig. 11.—Roentgenograms of patient who survived insertion of prosthetic valve.

the patient was being rewarmed, the heart spontaneously reverted to a regular rhythm at 32° C. Assisted circulation was continued for an additional 20 minutes. Thereafter, she maintained an adequate cardiac output with good systemic pressures. Aortic pulse tracings before operation were characteristic of aortic insufficiency and were normal after the valve was placed. The roentgenogram after operation shows the valve in place (Fig. 11, *C* and *D*).

During the first 3 postoperative days, the patient had premature ventricular beats and idioventricular foci alternating with sinus tachycardia. Adjustment of the digitalis dosage corrected this situation. During the ensuing 10 days she ran a low-grade fever to 102.4° F. and broad spectrum antibiotics were given. Blood cultures were negative. The remainder of

her course was uneventful. She has remained on digitalis and a low-sodium diet. Diuretics have not been necessary. Her condition is entirely satisfactory at an essentially normal activity level.

In spite of inadequate coronary perfusion this patient was successfully resuscitated. Survival appears to be correlated with a compact nondilated left ventricle.

CASE 3.—R. H. (PBBH No. 4P572), a 28-year-old man, had rheumatic fever at the age of 11. He was asymptomatic until age 24 when he developed acute dyspnea. Three months prior to this admission, the dyspnea and fatigue became refractory to medical therapy. At this time he was observed fluoroscopically and the cardiac and aortic activity were exaggerated, characteristic of aortic insufficiency. There was no angina or syncope.

Physical examination: The blood pressure was 160/60 mm. Hg. He had Corrigan pulses. There were expiratory râles. The point of maximal impulse was in the anterior axillary line and was accompanied by a characteristic left ventricular heave. The right ventricular contraction was not prominent. The aortic second sound was normal but the pulmonic sound was accentuated. At the base there was a Grade 2 rough systolic ejection murmur and a Grade 4 diastolic diminuendo murmur along the left sternal border. At the apex there was a Grade 3 pansystolic murmur and a Grade 2 diastolic rumble with an opening snap. In the tricuspid area there was a Grade 3 diffuse rough pansystolic murmur that was louder during inspiration. The liver edge was palpated two fingerbreadths below the right costal margin. There was no edema.

X-ray studies (Fig. 10, *B*) and fluoroscopy revealed considerable enlargement of the left ventricle (3+), right ventricle (2+), and left auricle. The transverse diameter was increased 34 per cent above the predicted normal and the pulmonary vascular bed was congested. There was no calcification and no rocking motion (although this had been vigorous).

Electrocardiogram revealed auricular fibrillation, intraventricular conduction defect, and left ventricular hypertrophy.

Combined heart catheterization revealed a total cardiac output of 6.2 L./min./M.^{2} with a regurgitant flow estimated at 2.91 L./min./M.^{2} or 47 per cent of total cardiac output. Pressures were elevated in all chambers of the right heart. Pulmonary artery pressure rose from a resting level of 61/30 to 110/58 mm. Hg with exercise indicating left ventricular failure. Left ventricular pressures were not obtained.

Hospital course: On bed rest and a full medical program, the patient lost 3 kilograms of fluid. At open operation, a caged-ball valve was inserted by the technique described. The patient was cooled to 28° C. Ventricular fibrillation occurred at 29° C. There was inadequate coronary perfusion during 56 minutes of aortic occlusion. The tenth defibrillating shock of 280 volts at 1/20 second restored regular rhythm and effective left ventricular contractions followed. Partial bypass was continued for an hour after release of the aortic clamp. After operation, the temperature rose to 105° F. and there was a tachycardia of 130 on the second postoperative day. His temperature was lowered to 95° to 96° F. and vasopressors were needed on the third day. The patient remained lucid and cooperative under these conditions. Urinary output ranged from 400 to 950 c.c. daily and cardiac rate varied from 130 to 160 per minute. The rhythm showed frequent changes from a supraventricular tachycardia, possibly flutter, to auricular fibrillation that constituted a problem in management. There was mild gastric bleeding. *Staphylococcus aureus* was cultured from the tracheobronchial tree but blood cultures were negative. On the seventh postoperative day, hypothermia and vasopressors were discontinued because of improvement but, as on previous occasions, each elevation of temperature was associated with ventricular tachycardia. On the eighth postoperative day the patient developed hypotension, ventricular tachycardia and fibrillation, and died.

Postmortem examination revealed that the prosthetic valve was well placed with patent coronary ostia. There were massive old and recent myocardial infarctions of the apex of the left ventricle. The empty heart weighed 980 grams. Although there was no evidence of coagulation necrosis it is felt that the ten electrical shocks contributed to the obvious old and recent massive infarctions as a cause of death.

CASE 4.—L. L. (PBBH No. 4P675), a 32-year-old man, had had scarlet fever at the age of 9. He was discharged from the Army because of rheumatic heart disease. Acute nephritis and polyarthritis occurred at 27. He then remained well until one month prior to hospitalization when he developed left ventricular failure, cough, dyspnea, orthopnea, pulmonary edema, and exertional precordial pain radiating down both arms.

Physical examination: Bisferiens carotid pulses and pistol shot femoral pulses were present. The lungs were clear. The liver edge was palpated two fingerbreadths below the costal margin. The blood pressure was 145/40 mm. Hg and the heart was in normal sinus rhythm. A diffuse left ventricular heave was felt in the 6th interspace at the anterior axillary line. Grade 3 aortic systolic and diastolic murmurs were heard.

Roentgenograms (Fig. 10, *C*) and fluoroscopy revealed aortic valve calcification and marked left ventricular enlargement. A definite rocking beat was present. The transverse cardiac diameter was 65 per cent above predicted normal.

The electrocardiogram showed normal sinus rhythm, left auricular enlargement, left ventricular hypertrophy, and an intraventricular block.

At transseptal cardiac catheterization there was no mitral or aortic gradient at a cardiac output of 4.3 liters. The left ventricular end-diastolic pressure was 29 mm. Hg, indicating left ventricular failure. Dye dilution curves indicated marked regurgitation.

Hospital course: Diuretics produced a weight loss of 4.8 kilograms. At open operation on bypass, free aortic regurgitation was found to be due to enlargement of the aortic base and shift of the mitral cusp. There was slight calcification and commissural fusion. The temperature was reduced to 26° C. with a heat exchanger. Ventricular fibrillation occurred at 28.5° C. Both coronary arteries were perfused at approximately 120 c.c. per minute total flow. The left ventricle was vented through a No. 38 plastic catheter. Placement of the caged-ball valve required 60 minutes. There were 22 ineffective shocks at 280 volts of 1/20 of a second. Standstill was ultimately produced after giving 2 Gm. of quinidine and a shock of 130 volts for 3.5 seconds. The inability to place this massive ventricle in standstill with standard equipment clarified the need for more powerful defibrillators.

At *autopsy* the valve was well placed. The empty heart weighed 1,120 grams and was dilated. There was an old myocardial infarction at the apex of the left ventricle. No evidence of coagulation type necrosis was found.

CASE 5.—N. R. (PBBH No. 4P740), a 49-year-old man, was first known to have heart disease at the age of 26, when a murmur was heard. He gave no history of acute rheumatic fever. His first symptom, an isolated episode of syncope, occurred at the age of 34 after exertion. Exertional dyspnea developed at 41 years of age and became progressive. Two years before admission he had transient right-sided weakness and aphasia. This was treated with anticoagulants. Marked deterioration began a year prior to hospitalization. An episode of fever and congestive failure suggested subacute bacterial endocarditis. Penicillin was given, although an organism was never isolated. On admission, he was dyspneic after climbing one half flight of stairs and had orthopnea. He did not have angina, hemoptysis, peripheral edema, or further episodes of syncope. A vigorous medical and diuretic program had resulted in a weight loss of 5 kilograms during the preceding 2 weeks.

Physical examination: Bisferiens carotid pulses and pistol shot femoral pulses were present. The blood pressure was 110/60 mm. Hg and the heart was in normal sinus rhythm. There were fine moist râles at bases of both lungs. There was a moderate dorsal kyphoscoliosis. The liver edge was palpated three fingerbreadths below the costal margin. A diffuse left ventricular heave was felt in the sixth interspace outside the anterior axillary line. A Grade 4 aortic systolic murmur and a Grade 2 aortic diastolic murmur were heard.

Roentgenograms (Fig. 10, *D*) and fluoroscopy revealed marked left ventricular enlargement, a tortuous ascending aorta, and heavy aortic valve calcification. A rocking beat was not present. The transverse cardiac diameter was 63 per cent above predicted normal.

The electrocardiogram showed normal sinus rhythm with left ventricular hypertrophy, first degree left bundle branch block, and first degree heart block.

At transseptal cardiac catheterization there was a 29 mm. Hg mean systolic gradient across the aortic valve at a flow of 3.7 L./min. Peak left ventricular systolic pressure was 141 mm. Hg and the end-diastolic was 31 mm. Hg, indicating left ventricular failure. Dye dilution curves indicated marked regurgitation.

Ventilatory studies showed a moderate reduction in all values: vital capacity, 2.34 L. (55 per cent of predicted); maximum breathing capacity, 78 L. (69 per cent of predicted); and timed vital capacity, 73 per cent in one second.

Serum electrolytes and blood constituents were within accepted normal limits.

Hospital course: After 22 days of intensive medical therapy, the liver edge had receded to the costal margin, the lungs cleared, and the patient lost 6 more kilograms of weight. At open operation, on bypass, aortic insufficiency was due to contraction of all leaflets and a perforation in the base of the left coronary cusp. The right and left coronary bearing cusps were fused and heavily calcified. The caged-ball valve was inserted as illustrated (see Figs. 6 and 7). Bilateral coronary perfusion averaged more than 150 c.c. per minute total flow at normal temperatures. A single reservoir made an estimate of individual artery perfusion impossible. The aorta was cross-clamped for approximately 60 minutes but at 35 minutes the ventricle fibrillated. During bypass, massive bleeding developed from all incised surfaces. This necessitated rapid replacement transfusions. Ventricular fibrillation was successfully converted to arrest by one shock of 250 volts at 0.15 second, but a sustained orderly contraction was not restored. Continued bleeding necessitated 43 units of citrated blood within 2 hours. Despite continued assisted circulation, resuscitative efforts failed. It was never possible to take this patient off of extracorporeal circulation.

In spite of apparently adequate coronary perfusion, ventricular fibrillation developed making evident the necessity for measured individual coronary artery perfusion. This patient's uncontrollable bleeding problem, requiring replacement transfusions with ACD blood, may have imposed additional metabolic burdens and complicated the resuscitative effort, even though there was the usual calcium coverage.

B. *Discussion of Cases.*—

In the first patient the immediate problem was inadequate venting or decompression of the left ventricle. In previous open-heart surgery, left auricle venting had been sufficient. However, with the valve in place, a reverse transaortic gradient was not maintained and the valve became incompetent. Pressure from the aorta was transmitted directly to the dilated left ventricle. This afforded hydraulic multiplication of the pump pressure so that the ventricle violently expanded. The venting cannula was then rearranged in subsequent procedures so as to pass through the wall of the left auricle and the mitral valve, directly into the left ventricle. Decompression could be controlled by aspiration from the roller pump or by gravity.

The second patient was cooled to 28° C. Partial coronary perfusion with imperfect apparatus was carried out. There was good left ventricular decompression through a No. 38 Bardic catheter.* The heart went into ventricular fibrillation at 29° C. Following valve insertion, the heart spontaneously reverted to a regular rhythm as the patient's temperature rose to 32° C. She has done extremely well since. Her roentgenograms are shown in Fig. 11. She does not have an extremely large ventricle. This may account for the success.

The third patient had a much larger ventricle (Fig. 10, *B*), both dilated and hypertrophied. In spite of adequate decompression of the left ventricle,

*C. R. Bard, Inc., Summit, N. J.

the heart fibrillated and defibrillation was not effective until the tenth shock of 280 volts at 1/20 sec. Thereafter he could maintain an effective blood pressure only with vasopressors and at a body temperature of 34° C. Whenever the temperature was brought to normothermic levels, tachycardia became excessive and ventricular irritability increased. After 7 days of such hypothermia and in the presence of a gradual increase in pulmonary infection yet general improvement, the patient was warmed. This was found incompatible with his heart function. He had cardiac irritability, ventricular fibrillation,

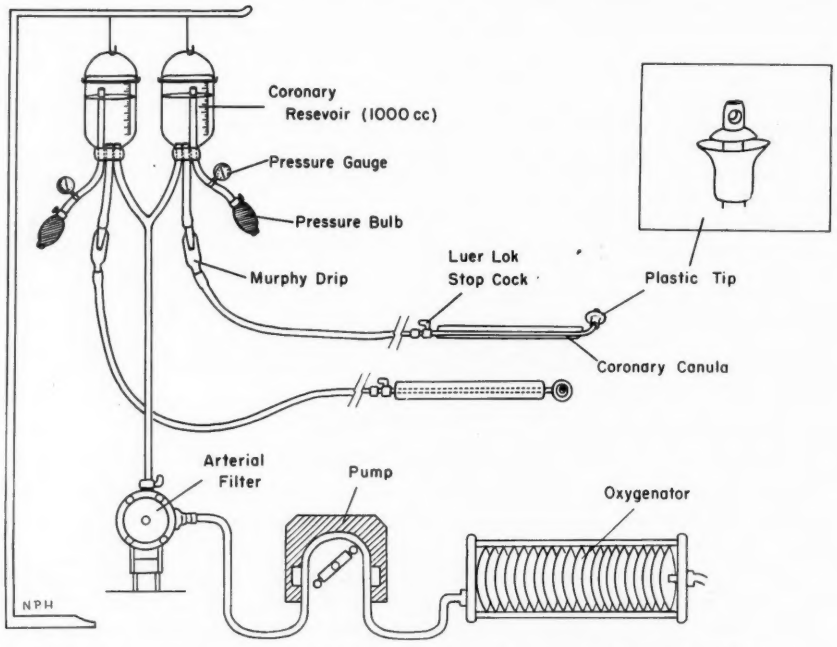

Fig. 12.—Coronary perfusion apparatus for separate perfusion of coronary arteries.

and succumbed on the eighth postoperative day. His difficulty at operation stemmed initially from ventricular fibrillation, then secondarily to the inability to defibrillate promptly. It was thought that coagulation damage had been caused by the multiple shocks but evidence on microscopic examination is lacking. He did, however, show a massive old and recent apical myocardial infarction. The old lesion antedated the operation. It is interesting and perhaps significant to know that a vigorous left ventricle and markedly active aortic arch had been observed in previous years, but on admission to the hospital the arch and ventricle were quiet. Presumably the infarction and failure accounted for this.

The fourth patient had a still larger ventricle (Fig. 10, *C*) although in a better state of compensation as attested by the end-diastolic pressure and by the vigor of the pulsation. This caged-ball valve was placed without difficulty.

Decompression was good but ventricular fibrillation refractory. Two hundred and eighty volts at 1/20 second did not cause standstill. It was not until the twenty-third shock, with another defibrillator delivering 130 volts for 3½ seconds, that this ventricle was arrested. However, a satisfactory beat was never restored. Although evidence of coagulation necrosis is not clear in the microscopic specimens of heart muscle, we find it difficult to believe that the 22 ineffective shocks of 280 volts were not related to this death. This patient demonstrated the necessity for better coronary perfusion and a more powerful defibrillator that could produce standstill in such a massive ventricle. Apparently these ventricles may have been so large as to render ordinary defibrillators inadequate.

Fig. 13.—Handles and tips of the coronary cannulas.

The coronary perfusion equipment had a common trunk of origin from the reservoir, so we could not tell how much of the blood was being delivered to each coronary artery. Furthermore we had not fully appreciated the importance of perfusing both coronary arteries. Contrary to the popular impression, Schlesinger[21] showed that in all but 18 per cent of people the right coronary artery supplies a significant part of the left ventricle or septum. This pointed up the necessity for effective perfusion of both arteries and independent reservoirs so that the flow to each could be verified.

The improved coronary perfusion apparatus (Fig. 12), handles and tips (Fig. 13), were then devised.* A defibrillator with a set time interval of 0.15 second and a variable manual time control delivering up to 250 volts was then obtained.†

*Manufactured by Davol Company, Providence, R. I.
†Electrodyne Company, Norwood, Mass.

In the fifth patient, the incision at the base of the aorta was extended to afford better exposure and perfusion of the coronary arteries (see Fig. 6). Even so, the heart did fibrillate. More effective shock brought this large ventricle into standstill followed by a transient organized beat, but this was neither effective nor sustained. However, this patient presumably died because of a bleeding problem. Forty-three citrated blood transfusions were given in approximately 2 hours, but the bleeding became steadily more profuse from all incised surfaces. This may have contributed to or caused the failure. Excess citrate or the alkalosis resulting from its oxidation are both hazardous in the digitalized cardiac surgical patient, despite the measures commonly used to prevent citrate "toxicity."[22]

COMMENT

At this point it is clear that this valve and the technique of placing it do not constitute a solution to the problem of aortic insufficiency in patients who have markedly dilated left ventricles in failure. The roentgenograms (Fig. 11) indicate that the only survivor with a complete prosthesis was a woman who had a smaller ventricle than those who died (Fig. 10).

We plan to continue this effort. The combination of better coronary perfusion, perhaps hypothermia at 32° C., improved left ventricle decompression, and adequate defibrillating apparatus may allow the necessary time (50 to 70 minutes) for valve installation.

So far we have not been able to use our synchronized pulsatile pump[23] effectively in this situation. It has been impossible to synchronize the pump inflow with the disorderly ventricular complex. Possibly this apparatus may be rendered useful by changing the activating impulse from the R wave of the electrocardiogram to the alterations in aortic pulse pressure.

This group of patients threatens to remain beyond surgical correction. On the other hand, if they can be salvaged by a suitable prosthesis, we can apply this knowledge to the treatment of combined aortic stenosis and regurgitation and, later, to pure stenosis. Better valves than the caged-ball valve may well be developed, but the site and technique of insertion described here seem valid and the testing equipment may continue to be useful.

SUMMARY

1. Anatomic and pathologic features of aortic insufficiency are reviewed.

2. The rationale for nonflexible cusp extension on the neutral flexible cusp is presented.

3. The clinical experience with 4 patients who had nonflexible prosthetic cusp extension in aortic insufficiency is mentioned.

4. The requirements for testing total aortic valve prostheses as to competence, resistance, fatigue, and trauma to blood elements are mentioned and certain testing equipment is described.

5. Special problems of decompressing (sumping) the left ventricle with reference to mural tension and ventricular dilatation are pointed out.

6. Shortcomings in conventional coronary perfusion equipment and defibrillation are considered and better equipment is described.

7. Consideration is given the problems encountered in four unsuccessful operations involving total aortic valve insertion in patients with free insufficiency with dilated left ventricles in failure.

8. A case of successful total aortic valve replacement with a caged-ball valve is reported. Future possibilities of this technique are considered.

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REFERENCES

1. Lillehei, C. W., DeWall, R. A., Gott, V. L., and Varco, R. L.: The Direct Vision Correction of Calcific Aortic Stenosis by Means of a Pump Oxygenator and Retrograde Coronary Sinus Perfusion, Dis. Chest **30**: 123-132, 1956.
2. Lillehei, C. W., Gott, V. L., DeWall, R. A., and Varco, R. L.: The Surgical Treatment of Stenotic or Regurgitant Lesions of the Mitral and Aortic Valves by Direct Vision Utilizing a Pump Oxygenator, J. THORACIC SURG. **35**: 154-189, 1958.
3. Bailey, C. P.: Discussion of paper by Harken et al.[4]
4. Harken, D. E., Black, H., Taylor, W. J., Thrower, W. B., and Soroff, H. S.: The Surgical Correction of Calcific Aortic Stenosis in Adults, J. THORACIC SURG. **36**: 759-776, 1958.
5. Harken, D. E., Black, H., Taylor, W. J., Thrower, W. B., Soroff, H. S., and Bush, V.: The Surgical Correction of Calcific Aortic Stenosis in Adults: Technique of Transaortic Valvuloplasty, Cardiology **4**: 135-146, 1959.
6. Brock, R.: Surgical Treatment of Aortic Stenosis, Brit. M. J. **1**: 1019-1028, 1957.
7. Glover, R. P., and Gadboys, H. L.: Seven Years' Experience With Transventricular Aortic Commissurotomy, J. THORACIC SURG. **36**: 839-856, 1958.
8. Swan, H., Wilkinson, R. H., and Blount, S. G., Jr.: Visual Repair of Congenital Aortic Stenosis During Hypothermia, J. THORACIC SURG. **35**: 139, 1958.
9. Morrow, A. G.: Personal communication regarding the open correction of aortic stenosis under hypothermia.
10. Wells, R. E., Taylor, W. J., Black, H., and Harken, D. E.: Pulmonary Function in the Selection of Patients for Open Heart Surgery, Dis. Chest. (In press.)
11. Harken, D. E., Black, H., Taylor, W. J., Thrower, W. B., and Ellis, L. B.: Reoperation for Mitral Stenosis: A Discussion of Postoperative Deterioration and Methods of Improving Initial and Secondary Operations, Circulation. (In press.)
12. Taylor, W. J., Thrower, W. B., Black, H., and Harken, D. E.: The Surgical Correction of Aortic Insufficiency by Circumclusion, J. THORACIC SURG. **35**: 192-205, 230-231, 1958.
13. Garamella, J. J., Andersen, J. G., and Oropeza, R.: The Surgical Treatment of Aortic Insufficiency by Open Plastic Revision of the Tricuspid Aortic Valve to a Bicuspid Valve, Surg. Gynec. & Obst. **106**: 679-686, 1958.
14. Garamella, J. J., Andersen, J. G., Oropeza, R., Veloso, A., and Naidu, R.: The Surgical Treatment of Aorta Insufficiency by Conversion of Tricuspid Aortic Valve to a Bicuspid Valve, J. THORACIC SURG. **37**: 177, 1959.
15. Bailey, C. P., and Zimmerman, J.: The Surgical Correction of Aortic Regurgitation—Bicuspid Conversion, Am. J. Cardiol. **3**: 6-21, 1959.
16. Muller, W. H., Jr., Warren, W. D., Dammann, J. F., Jr., Beckwith, J. R., and Wood, J. E., Jr.: Surgical Relief of Aortic Insufficiency by Direct Operation on the Aortic Valve, Circulation **21**: 587-597, 1960.
17. Bahnson, H.: Personal communication.
18. Harken, D. E., Black, H., Ellis, L. B., and Dexter, L.: The Surgical Correction of Mitral Insufficiency, J. THORACIC SURG. **28**: 604-627, 1954.

19. Hufnagel, C. A., Harvey, W. P., Rabil, P. J., and McDermott, R. F.: Surgical Correction of Aortic Insufficiency, Surgery 35: 673, 1954.
20. Hufnagel, C. A., Vilkgas, P. D., and Nahas, H.: Experience With New Types of Aortic Valvular Prostheses, Ann. Surg. 147: 636-645, 1958.
21. Schlesinger, M. J.: Relation of Anatomic Pattern to Pathological Conditions of the Coronary Arteries, Arch. Path. 30: 403-415, 1940.
22. Moore, F. D.: Metabolic Care of the Surgical Patient, Philadelphia, 1959, W. B. Saunders Company.
23. Clauss, R. H., Birtwell, W. C., Albertal, G. A., Lunzer, S., Taylor, W. J., Fosberg, A. M., and Harken, D. E.: Assisted Circulation. I: The Arterial Counter Pulsator, J. THORACIC SURG. (In press.)

(For Discussion, see page 799.)

SURGICAL TREATMENT OF VENTRICULAR SEPTAL DEFECT

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PATIENTS with severe pulmonary hypertension and infants pose the greatest challenges in the surgical treatment of ventricular septal defect. In other circumstances good results have been obtained with relative ease. The experience here reported indicates that, by application of proper techniques, a low hospital mortality can be achieved in infants as well as in patients with most types of ventricular septal defect with severe pulmonary hypertension.

MATERIAL

All 320 cases in which ventricular septal defect was treated on one surgical service at the Mayo Clinic prior to April 1, 1960, are included in this study. Results in the total group operated upon in this clinic are similar, but their inclusion would have introduced additional variations in technique and management, making analysis more difficult. The history, operative reports, chest roentgenograms, electrocardiograms, and necropsy findings from each case were reviewed individually.

Cases of tetralogy of Fallot, as previously defined,[1] have not been included. Likewise, cases in which the interventricular communications were part of the malformations known as complete common atrioventricular canal, single ventricle, origin of both vessels from right ventricle, infundibular pulmonary stenosis with tiny ventricular septal defect, or corrected transposition of the great vessels have been excluded.

Associated malformations other than pulmonary stenosis are listed in Table I. Pulmonary stenosis is not included in the table, since patients with this entity are categorized separately in the section on results. The lesions indicated were corrected at the time of repair without complication, except for severe mitral and aortic valvular insufficiency and the combination of patent ductus arteriosus and coarctation of the aorta. These also will be considered separately

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under results. The shunt across a high ventricular septal defect was recognized as being wholly or in part into the right atrium in 20 cases. In 15 of these, pulmonary hypertension was only mild or moderate.

TABLE. I. CERTAIN ASSOCIATED CARDIOVASCULAR ANOMALIES IN 320 CASES WITH OPERATION FOR VENTRICULAR SEPTAL DEFECT

ANOMALIES	CASES
Repaired at Time of VSD Repair	
Atrial septal defect	20
Patent ductus arteriosus	16
Aortic-pulmonary fistula	1
Aortic-valve insufficiency	5
Mitral-valve lesions	
Stenosis	1
Insufficiency,* severe	5
Coarctation of aorta and patent ductus arteriosus	4
Not Repaired at Time of VSD Repair	
Aortic-valve deformity with minimal stenosis	3
Mitral-valve insufficiency,* mild	9
Anomalous right coronary artery supplying left anterior descending artery†	4
Coarctation of aorta	4
Persistent left superior vena cava	22
Stenosis of right or left pulmonary artery	2
Absence of left pulmonary artery	1
Right aortic arch	2

*Exclusive of endocardial-cushion defects.
†Preserved without damage in all cases.

Patients were categorized as having mild pulmonary hypertension when pulmonary-artery systolic pressure was 45 per cent or less of systemic-artery systolic pressure, moderate pulmonary hypertension when it was between 45 per cent and 75 per cent of systemic-artery pressure, and severe pulmonary hypertension when it was 75 per cent or more of systemic-artery pressure. Patients with severe pulmonary-artery hypertension were further divided into those whose pulmonary/systemic blood flow ratios were high (1.8 or more), moderately elevated (1.3 to 1.8), and low (less than 1.3). When complete data had been obtained by cardiac catheterization without anesthesia, they were used for classification of the case. In some instances, particularly among small children, the conditions existing during cardiac catheterization result in hypoventilation. Values for pulmonary-artery pressure then tend to be abnormally high, and those for pulmonary/systemic flow abnormally low. This can make a patient appear less favorable for operation than in fact he is. In such instances, the patient was categorized according to the clinical data and the pressure measurements which were made routinely at the time of surgery. These findings alone were utilized in cases where preoperative catheterization data were not available. The clinical data were derived from physical examination, the chest roentgenogram, and the electrocardiogram. For the most part, criteria used for categorization of the patient on the basis of these data have been validated previously.[2-6]

Defects immediately below the aortic leaflets are called *high ventricular septal defects.* Although they vary considerably in their exact relation to the

tricuspid-valve leaflets and in their size, basically similar techniques of repair can be used for all. With some high defects, a small muscular defect was associated. A special category of high defects are those immediately beneath the commissure between right and left aortic cusps, and thus *immediately beneath the pulmonary valve.* Certain defects not immediately beneath the aortic leaflets are triangular, with their cephalad margin formed by the tricuspid valve. Frequently some of the chordae tendineae of this valve are attached to a papillary muscle within the left ventricle. Very often there is a cleft in the anterior mitral leaflet alone or in combination with one in the septal leaflet of the tricuspid valve. These are believed to be *endocardial-cushion defects. Defects in the muscular septum* are removed from both aortic and tricuspid valves. When three or more such defects are found, the septum may be in fact multifenestrated, and this situation is designated *"Swiss cheese"* septum.

When the repair is adjudged complete by postoperative cardiac catheterization or careful clinical study, the ventricular septum is termed *closed.* In cases where postoperative cardiac catheterization has not yet been done, but the clinical findings are suggestive although not indicative of residual shunt, the result is termed *indeterminate.* The septum is termed *open* when clinical or catheterization data indicate the presence of a shunt after operation.

After careful review of all the pertinent data and with realization that absolute accuracy is not possible, a cause was assigned for each death (Table II). Several of these causes have been commented upon previously.[7]

METHODS AND SURGICAL TECHNIQUE

Operation was advised for all patients with severe pulmonary hypertension whose pulmonary/systemic blood flow ratio was thought to be greater than 1.[2] Fifty per cent of patients in the series were from this group. Patients having ventricular septal defect without pulmonary hypertension, without evidence of cardiac enlargement or hyperemia of the lungs in the chest roentgenogram, and without electrocardiographic abnormality were not operated upon.

A Gibbon-Mayo pump-oxygenator was used in all cases. Since the fall of 1958, a modified apparatus incorporating reduced priming volume and mechanical filming of the oxygenator has been employed in operations on infants and small children. Some of the characteristics of perfusions in the early experience have been described.[7, 8] In the last 2 years, flow rates have been 2.2 L. per minute per square meter (for adults) to 2.4 L. per minute per square meter (for children), and temperature has been maintained at 37° C. by the use of a Brown-Emmons heat exchanger.[9]

Anesthetic and postoperative management have been described.[10, 11] Recent improvements include the use of halothane (Fluothane) as the anesthetic agent,[12] reliance upon systemic venous and arterial blood pressures rather than blood balance figures as guides to administration of blood, and more vigorous seeking out and treatment of metabolic acidosis in patients having low cardiac output

TABLE II. CAUSES OF HOSPITAL DEATH IN 320 CASES WITH OPERATION FOR VENTRICULAR SEPTAL DEFECT

	1955	1956	1957	1958	1959
Group 1					
Acute early pulmonary complications	6[a]	2[b]			
Unexpected sudden death	3[b]	3[b]			
Respiratory failure (apnea)		2[b]		1[b]	
Pseudomonas subacute bacterial endocarditis		1			
Severe hyperthermia, hypotension (? blood reaction)					1
Group 2					
Inadequate repair of mitral-valve insufficiency			1[c]		
Inadequate repair of aortic-valve insufficiency					1
Failure in management of associated coarctation and patent ductus arteriosus					2
Failure in repair of ventricular septal defect			1[c]	1[c]	1[cd]
Digitalis intoxication precipitating ventricular fibrillation					2[d]
Sudden death sixth postoperative day during removal of ECG electrodes					1
Low cardiac output, poorly treated	1[cc]	2[c]	3[c]	4[c]	2[cf]
Group 3					
Complete heart block		5[cg]	5[h]	1[i]	
Severe early tachycardia		2		2[b]	
Late (2 weeks) sudden tachycardia and death		1[j]	1[k]		
Group 4					
Chronic late pulmonary complications				2[l]	
Low cardiac output, well treated					2[cf]
Low cardiac output from acute right ventricular failure in operating room					1[c]
Totals	9	17	9	10	12

a–Five had severe pulmonary hypertension. Two, including 1 with moderate pulmonary hypertension, were infants.
b–All were infants.
c–One included elsewhere also.
d–Endocardial-cushion defects.
e–All had severe pulmonary hypertension.
f–Endocardial-cushion or "Swiss cheese" defects.
g–Four had severe pulmonary hypertension; 1 had pulmonary stenosis.
h–Four were not driven immediately; 1 died in asystole in spite of pacemaker functioning since surgery.
i–Endocardial-cushion defect. Pacemaker functioning since surgery.
j–Endocardial-cushion defect. Death preceded by paroxysmal nodal tachycardia.
k–Multiple ventricular premature contractions, preoperatively and postoperatively.
l–Both infants with intractable pneumonia and atelectasis preoperatively.

after operation.[13] A median sternotomy incision has been employed since October, 1958. Since August, 1956, cardiac asystole has been used routinely, induced at first by potassium citrate and, since January, 1959, by ischemia.[14] Only a few defects were repaired through the right atrial approach.

Heart block was variously treated during this experience (Fig. 1). The method used during the past 2 years has been detailed previously.[15] Tracheostomy was employed occasionally in 1955 and early 1956, but rarely since.

A number of techniques, with and without a prosthesis, were employed in the first 3 years in an attempt to obtain complete closures of the ventricular septal defects.[16, 17] In 1958 and 1959, all high defects were repaired by direct suture with the stitches placed in a manner designed to avoid the bundle of His (Fig. 1). The line of closure is transverse to the axis of the outflow tract of the left ventricle. Interrupted sutures are placed from the posterior angle forward. Posterior to the papillary muscle of the conus, stitches are kept well

away from the free edge of the septum, since it is in this area and at the posterior angle that damage to the bundle of His is most apt to occur. Along the cephalad edge of the defect the stitches actually bite firmly into the root of the aorta, just missing the aortic leaflets, which must be clearly visualized. Many defects in their anterior portion veer downward away from the aortic leaflets and here the stitches are placed only in the muscle. An assistant, by traction on the aortic clamp, brings the aortic root down against the septum as the stitches are being tied under precisely the proper tension.

Defects immediately beneath the pulmonary valve are usually somewhat triangular. Unless the defect is small, a prosthesis of Teflon is used. Endocardial-cushion defects are characteristically triangular and require the use of a prosthesis. All muscular defects have been repaired by direct suture.

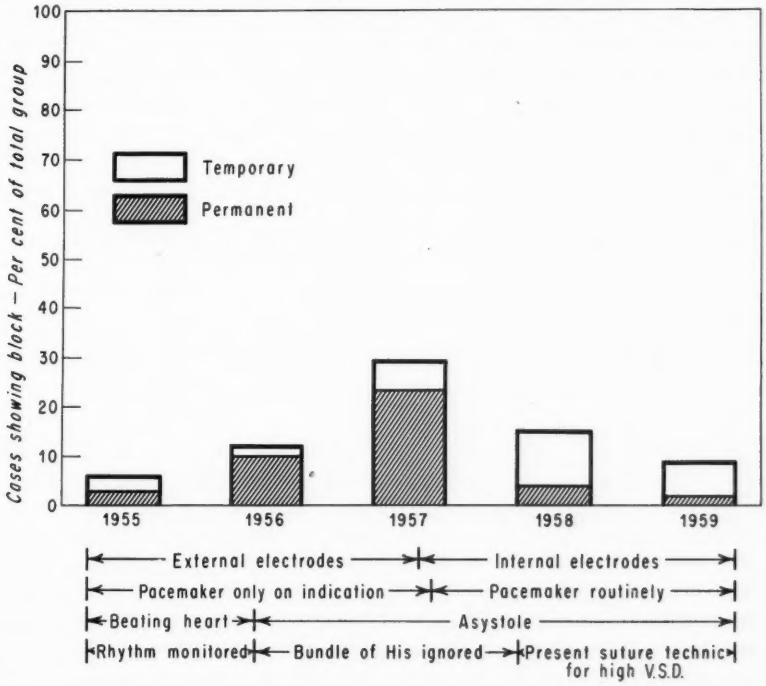

Fig. 1.—Incidence of heart block and pertinent items of management following repair of ventricular septal defect.

RESULTS

Hospital Mortality.—The mortality was high—43 per cent—among the 14 patients with certain additional serious cardiovascular anomalies (Table III). The combination of patent ductus arteriosus, coarctation of the aorta, and ventricular septal defect was not recognized preoperatively in 3 of the 4 patients in whom it occurred, and hence caused an especially high mortality in this group. In order to isolate the risk of surgery specifically for ventricular septal

defect, the 14 patients with these particular associated anomalies have been excluded from the further observations on mortality.

TABLE III. HOSPITAL MORTALITY WITH OPERATION FOR VENTRICULAR SEPTAL DEFECT IN PATIENTS HAVING CERTAIN ASSOCIATED ANOMALIES

ASSOCIATED ANOMALIES	1957*		1958		1959		TOTAL	
	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES	DEATHS	CASES
Severe mitral-valve incompetence	2	3	–	1	–	1	2	5
Severe aortic-valve incompetence	–	–	–	1	1	4	1	5
Patent ductus arteriosus and coarctation	–	1†	–	–	3	3‡	3	4

*In all tables and in the text, the period designated by a year extends from April 1 of that year to April 1 of the following year.

†Coarctation mild.

‡In 1, patent ductus arteriosus and coarctation corrected one year previously.

In the remaining group of 306 patients with ventricular septal defect (Table IV), the hospital mortality had dropped to 9 per cent in 1959. Particularly striking in that year is the rate of 6 per cent among the 18 patients 2 years of age or younger. Eight of these were infants, less than 1 year of age, in severe cardiac failure. The one death was in such an infant who at surgery was found to have an endocardial-cushion type of ventricular septal defect.

TABLE IV. HOSPITAL MORTALITY FOLLOWING REPAIR OF VENTRICULAR SEPTAL DEFECT (306 CASES), RELATED TO AGE

AGE (YR.)	1955			1956			1957			1958			1959		
	HOSP. DEATH		TOTAL CASES	HOSP. DEATH		TOTAL CASES	HOSP. DEATH		6 TOTAL CASES	HOSP. DEATH		TOTAL CASES	HOSP. DEATH		TOTAL CASES
	NO.	%		NO.	%		NO.	%		NO.	%		NO.	%	
0–2	5	63	8	8	67	12	–	0	7*	6	35	17	1	6	18
3–4	1	25	4	–	0	10	2	14	14	3	20	15	3	19	16
5–7	1	17	6	5	42	12	1	5	20	–	0	18	2	9	23
8–14	2	15	13	1	9	11	3	21	14	1	6	16	2	10	21
> 14	–	0	2	3	60	5	1	20	5	–	0	11	0	0	8
Totals	9	27	33	17	34	50	7	12	60	10	13	77	8	9	86

*No infants under 1 year of age.

Patients with mild or moderate pulmonary-artery hypertension, as well as those with pulmonary stenosis, have been operated on with a reasonable mortality throughout this experience (Table V). In the last 2 years, among the 76 patients in these categories, the only deaths occurred in 2 infants in cardiac failure operated upon in 1958. Patients with severe pulmonary-artery hypertension, as a group, had a significant hospital mortality rate, although it was reduced to 24 per cent in 1958 and 20 per cent in 1959. The pulmonary systemic blood flow ratio does appear to affect the risk of operation, since particularly in the last 2 years the risk was low where this ratio was high.

The "Swiss cheese" type of defect and endocardial-cushion defects have been associated with a higher hospital mortality than the other types (Table VI). In 1959, the risk of surgery was only 4 per cent in cases of high ventricular septal defect with severe pulmonary hypertension.

Causes of Death (Table II).—Complications considered to be the result of the perfusion technique, those in Group 1, caused most of the deaths in 1955 and 1956, but almost none thereafter. Acute early pulmonary complications disappeared by 1957. Tracheostomy was necessary occasionally in 1955 and 1956, but has been used in only 3 cases in the last 3 years. Unexpected sudden death and respiratory failure occurred in 8 patients—all infants—in 1955 and 1956. The low hospital mortality in infants in the last 2 years was achieved in large part by the near elimination of these types of deaths. The absence of renal or gross cerebral complications from the entire series is noteworthy.

The deaths tabulated in Group 2 represent inadequate surgical procedures or improper postoperative care. Group 3 includes those deaths which were due to disturbances of cardiac rhythm. In Group 4, all patients dying of low cardiac output had severe pulmonary hypertension. The patient who died in the operating room of acute right ventricular failure, manifested by falling left atrial and systemic arterial pressures and rising right atrial pressure, had identical pulmonary-artery and aortic pressures after repair of the defect as well as prior to it.

Table II again indicates that, in all years, patients who either had severe pulmonary hypertension or were of very small size were the most likely to have fatal complications. Thus, the hospital mortality in 1955 and 1956, when operative and perfusion inadequacies definitely were present, would have been lower if both infants and all patients with severe pulmonary hypertension had been denied operation.

Heart Block.—The incidence of heart block is depicted in Fig. 1, along with pertinent matters of technique. With the technique presently used, the incidence of permanent heart block was 4 per cent in 1958 and 2 per cent in 1959.

Six of the 8 patients in whom temporary or permanent heart block developed during 1955 and 1956 died in the hospital. Use of an internal electrode and routine driving of the heart with an electrical pacemaker[18] reduced the hospital mortality significantly, with 12 deaths among the 38 patients having temporary or permanent heart block in 1957, 1958, and 1959. Yet the continuing serious import of permanent heart block is indicated by the fact that although in 1957 9 patients with complete heart block left the hospital alive, 4 of them died some months later from Stokes-Adams attacks.

There were no instances of heart block after repair of a muscular defect or a defect immediately beneath the pulmonary-valve ring. Block occurred in 1 patient with a "Swiss cheese" defect, and in 3 of the 11 patients with endocardial-cushion defect.

Completeness of Repair.—Regrettably, in 1955, one third of the patients had an open ventricular septum after operation (Table VII). This resulted from inexperience in methods of exposure within the right ventricle, haste during the repair because of doubts as to the safety of perfusion, uncertainty as

TABLE V. MORTALITY FOLLOWING REPAIR OF VENTRICULAR SEPTAL DEFECT

PULMONARY-ARTERY HYPERTENSION	1955			1956			1957			1958			1959		
	HOSP. DEATH NO.	HOSP. DEATH %	TOTAL CASES	HOSP. DEATH NO.	HOSP. DEATH %	TOTAL CASES	HOSP. DEATH NO.	HOSP. DEATH %	TOTAL CASES	HOSP. DEATH NO.	HOSP. DEATH %	TOTAL CASES	HOSP. DEATH NO.	HOSP. DEATH %	TOTAL CASES
Mild	–	0	7	1	9	11	–	0	6	–	0	17	–	0	14
Moderate	2*	50	4	–	0	7	1	5	20	2*	10	21	–	0	20
Severe	7	33	21	14	47	30	5	17	29	8	24	33	8	20	41
High $\frac{\text{pulm.}}{\text{sysm.}}$ flow ratio	4	24	17	14	52	27	2	12	17	4	21	19	2	10	21
Moderate $\frac{\text{pulm.}}{\text{sysm.}}$ flow ratio	2	100	2	–	0	2	3	25	12	3	23	13	4	25	16
Low $\frac{\text{pulm.}}{\text{sysm.}}$ flow ratio	1	50	2	–	0	1	–	0	–	1	100	1	2	50	4
Mild, with pulmonary stenosis	–	0	1	2	100	2	1	20	5	–	0	6	–	0	11
Totals	9	27	33	17	34	50	7	12	60	10	13	77	8	9	86

*Both were infants.

TABLE VI. MORTALITY IN VENTRICULAR SEPTAL DEFECT ACCORDING TO ANATOMIC TYPE OF VENTRICULAR SEPTAL DEFECT

PULMONARY HYPERTENSION	TYPE OF DEFECT	1955			1956			1957			1958			1959		
		HOSP. DEATH		TOTAL	HOSP. DEATH		TOTAL	HOSP. DEATH		TOTAL	HOSP. DEATH		TOTAL	HOSP. DEATH		TOTAL
		NO.	%	CASES	NO.	%	CASES	NO.	%	CASES	NO.	%	CASES	NO.	%	CASES
Mild	High	–	0	7	1	11	9	–	0	6	–	0	16	–	0	13
	Beneath pulm. valve	–	–	–	–	–	–	–	–	–	–	–	–	–	0	1
	Muscular	–	–	–	–	0	1	–	–	–	–	0	1	–	–	–
	"Swiss cheese"	–	–	–	–	0	1	–	–	–	–	–	–	–	–	–
	Endocardial cushion	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Moderate	High	2*	50	4	–	0	7	1	6	17	2*	12	17	–	0	18
	Beneath pulm. valve	–	–	–	–	–	–	–	–	–	–	0	1	–	0	2
	Muscular	–	–	–	–	–	–	–	0	2	–	0	3	–	–	–
	"Swiss cheese"	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
	Endocardial cushion	–	–	–	–	–	–	–	0	1	–	–	–	–	–	–
Severe	High	5	28	18	9	39	23	3	13	24	6	23	26	1	4	27
	Beneath pulm. valve	1	50	2	1	100	1	1	100	1	–	0	2	1	33	3
	Muscular	–	–	–	1	100	1	–	0	1	–	0	3	–	0	3
	"Swiss cheese"	1	100	1	1	33	3	1	33	3	–	–	–	2	100	2
	Endocardial cushion	–	–	–	2	100	2	–	–	–	2	100	2	4	67	6
Pulmonary stenosis	High	–	0	1	2	100	2	1	20	5	–	0	5	–	0	10
	Muscular	–	–	–	–	–	–	–	–	–	–	0	1	–	0	1
Totals		9	27	33	17	34	50	7	12	60	10	13	77	8	9	86

*Infants.

TABLE VII. STATUS OF VENTRICULAR SEPTUM AFTER OPERATION FOR VENTRICULAR SEPTAL DEFECT

TYPE OF VENTRICULAR SEPTAL DEFECT	1955					1956					1957					1958					1959				
	OPEN		INDETERMINATE		TOTAL CASES	OPEN		INDETERMINATE		TOTAL CASES	OPEN		INDETERMINATE		TOTAL CASES	OPEN		INDETERMINATE		TOTAL CASES	OPEN		INDETERMINATE		TOTAL CASES
	NO.	%	NO.	%		NO.	%	NO.	%		NO.	%	NO.	%		NO.	%	NO.	%		NO.	%	NO.	%	
High	9	32	3	6	30	6*	16	1	2	41	2†	4	2	4	55	7	11	2	3	66	2†	3	4	5	76
Beneath pulm. valve	2	100	–	–	2	1‡	100	–	–	1	–	–	–	–	1	–	–	–	–	3	–	–	–	–	5
Muscular	–	–	–	–	0	1	50	–	–	2	–	–	–	–	4	–	–	–	–	8	–	–	–	–	5
"Swiss cheese"	–	–	–	–	1	2†	50	–	–	4	1§	33	–	–	3	–	–	–	–	0	1§	50	–	–	2
Endocardial cushion	–	–	–	–	0	–	–	–	–	2	–	–	–	–	1	1	50	–	–	2	1	17	1	17	6
Totals	11	33	3	9	33	10	20	1	2	50	3	5	2	3	64	8	10	2	3	79	4	4	5	5	94

*Only small L→R shunt in 3 of these 6.
†Only small L→R shunt in 1 of these 2.
‡Following subacute bacterial endocarditis.
§Only small L→R shunt in 1.

to exact pathologic anatomy in many instances, and lack of knowledge concerning proper techniques for closure of the defect. When insertion of a stitch produced atrioventricular dissociation, it was removed. Reluctance to replace it in that position frequently resulted in incompleteness of repair.

The adoption of induced asystole in August, 1956, provided better exposure within the heart. Confidence as to the safety of perfusion allowed more deliberate intracardiac surgery. The technique of repair was directed solely toward obtaining complete closure of the defect. Since knowledge of the cause of heart block was incomplete at that time, no attempt was made to avoid the region occupied by the bundle of His. Consequently, the incidence of open septum among patients operated upon in 1957 was reduced to 5 per cent, but the incidence of permanent heart block rose sharply to 23 per cent.

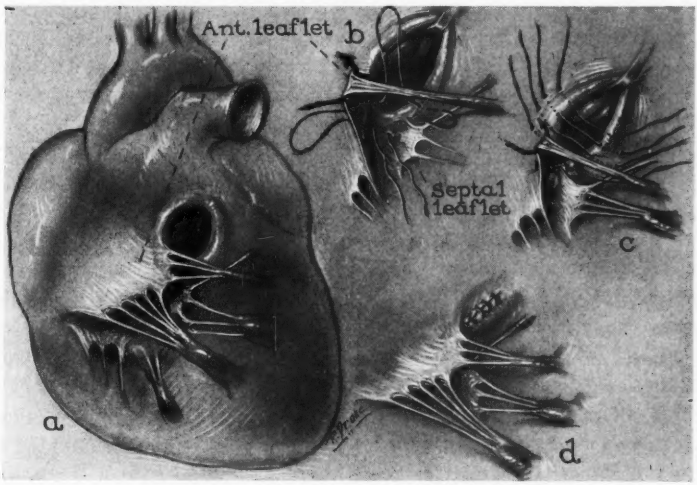

Fig. 2.—Technique of repair of high ventricular septal defect. *a*, Anatomic situation. *b*, A small retractor elevates the anterior corner of the defect. The first stitch is placed posteriorly in the manner shown, with care to avoid the free edge of the septum posterior to the papillary muscle of the conus. *c*, After all stitches are placed, the posterior one is tied, the knot lying on the right atrial aspect of the base of the tricuspid leaflets. *d*, No interference with tricuspid function is produced. No chordae tendineae are severed and no incision is made in the tricuspid leaflets.

This necessitated the change to the technique of repair used in 1958 and 1959 (Fig. 2). Probably because stitches near the posterior angle of the high ventricular septal defects were improperly located or too shallow, the incidence of open septum after operation rose to 10 per cent in 1958. In 1959, the incidence of known incomplete repair was reduced to 4 per cent. Since in this experience the presence of an open septum has always become manifest soon after operation, it is not feared that a longer follow-up will appreciably alter this figure.

The "Swiss cheese" type of defect, by its nature, continues to be sometimes difficult of total repair. Proper understanding of the anatomy and methods of exposure and repair of the endocardial-cushion type of ventricular septal defect have only recently been acquired.

COMMENT

Good results at a low hospital mortality have been obtained throughout this experience in children and adults having mild or moderate pulmonary hypertension. A similar experience is reported by others.[19, 20] Improved knowledge and techniques, gained through the investigations and experiences of others and ourselves, were required to achieve good results in infants and in patients with severe pulmonary hypertension.

The avoidance of deleterious effects from perfusion is probably more difficult in infants than in large patients. High flow rates were employed in the last 2 years, and use of substances such as antifoam was eliminated. The possibility of gaseous emboli was minimized by careful control of the temperature of blood before and during perfusion and by an alteration of the intracardiac sucker system. Use of a mechanical filmer for the oxygenator eliminated the hemodilution associated with the previously used saline filming technique. Maintenance of anesthesia by Fluothane, rather than by intravenously administered agents supplemented by relaxants, assured rapid recovery of the infant from the effects of anesthesia.

Good results in patients with severe pulmonary hypertension require a precise surgical technique and careful management after operation. The closure of high defects by direct suture, as practiced in the last 2 years, has resulted in a high incidence of complete repairs and a low incidence of heart block. Induced asystole provides excellent exposure, and has not seemed to add significantly to the risk of the operation. In the last few years, patients in this category usually have had an uncomplicated course. Occasionally there is evidence of low cardiac output early after perfusion, and failure to treat it vigorously resulted in some deaths. Maintenance of proper blood volume, prompt action against the metabolic acidosis which usually becomes marked under these circumstances, and digitalization, when required, have virtually eliminated this as a cause of death in our recent experience.

SUMMARY

An analysis is presented of 320 consecutive cases of ventricular septal defect in which operation was performed at the Mayo Clinic. A low hospital mortality and good results were achieved throughout the 5-year experience in older patients with mild or moderate pulmonary hypertension. Early, the mortality was high among infants and among all patients with severe pulmonary hypertension. Refinements in techniques of perfusion, operation, and management produced markedly improved results in these groups.

REFERENCES

1. Kirklin, J. W., Ellis, F. H., Jr., McGoon, D. C., DuShane, J. W., and Swan, H. J. C.: Surgical Treatment for the Tetralogy of Fallot by Open Intracardiac Repair, J. THORACIC SURG. 37: 22-46, 1959.
2. DuShane, J. W., and Kirklin, J. W.: Selection for Surgery of Patients With Ventricular Septal Defect and Pulmonary Hypertension, Circulation 21: 13-20, 1960.

3. DuShane, J. W., Weidman, W. H., Brandenburg, R. O., and Kirklin, J. W.: The Electrocardiogram in Children With Ventricular Septal Defect and Severe Pulmonary Hypertension: Correlation With Response of Pulmonary Arterial Pressure to Surgical Repair, Circulation **21**: 49-54, 1960.
4. Vickers, C. W., Kincaid, O. W., DuShane, J. W., and Kirklin, J. W.: Ventricular Septal Defect and Severe Pulmonary Hypertension: Radiologic Considerations in Selection of Patients for Surgery, Radiology **75**: 69-79, 1960.
5. Ziegler, R. F.: Electrocardiographic Studies in Normal Infants and Children, Springfield, Ill., 1951, Charles C Thomas, publisher, 207 pp.
6. Cayler, G. G., Ongley, P., and Nadas, A. S.: Relation of Systolic Pressure in the Right Ventricle to the Electrocardiogram: A Study of Patients With Pulmonary Stenosis and Intact Ventricular Septum, New England J. Med. **258**: 979-982, 1958.
7. Kirklin, J. W., McGoon, D. C., Patrick, R. T., and Theye, R. A.: What Is Adequate Perfusion? *in* U. S. National Institutes of Health, Surgery Study Section: Extracorporeal Circulation, Springfield, Ill., 1958, Charles C Thomas, publisher, pp. 125-138.
8. Moffitt, E. A., Patrick, R. T., Swan, H. J. C., and Donald, D. E.: A Study of Blood Flow, Venous Blood Oxygen Saturation, Blood Pressure, and Peripheral Resistance During Total Body Perfusion, Anesthesiology **20**: 18-26, 1959.
9. Brown, I. W., Jr., Smith, W. W., and Emmons, W. O.: An Efficient Blood Heat Exchanger for Use With Extracorporeal Circulation, Surgery **44**: 372-377, 1958.
10. Moffitt, E. A., and Theye, R. A.: Management of Anaesthesia, Perfusion and Supportive Care During Open Intracardiac Operations and Extracorporeal Circulation, Brit. J. Anaesth. **31**: 411-416, 1959.
11. Lyons, W. S., DuShane, J. W., and Kirklin, J. W.: Postoperative Care After Whole-Body Perfusion and Open Intracardiac Operations, J. A. M. A. **173**: 625-630, 1960.
12. Dawson, B., Theye, R. A., and Kirklin, J. W.: Halothane in Open Cardiac Operations: A Technic for Use With Extracorporeal Circulation, Anesth. & Analg. **39**: 59-63, 1960.
13. Ito, I., Faulkner, W. R., and Kolff, W. J.: Metabolic Acidosis and Its Correction in Patients Undergoing Open-Heart Operation, Cleveland Clin. Quart. **24**: 193-203, 1957.
14. Crafoord, C., Norberg, B., and Senning, Å.: Clinical Studies in Extracorporeal Circulation With a Heart-Lung Machine, Acta chir. scandinav. **112**: 220-245, 1957.
15. Lauer, R. M., Ongley, P. A., DuShane, J. W., and Kirklin, J. W.: Heart Block Occurring After Repair of Ventricular Septal Defect in Children, Circulation (In press).
16. Kirklin, J. W., Harshbarger, H. G., Donald, D. E., and Edwards, J. E.: Surgical Correction of Ventricular Septal Defect: Anatomic and Technical Considerations, J. THORACIC SURG. **33**: 45-57, 1957.
17. Kirklin, J. W., and McGoon, D. C.: Surgical Technique for Repair of High Ventricular Septal Defects, J. THORACIC SURG. **35**: 584-590, 1958.
18. Weirich, W. L., Gott, V. L., and Lillehei, C. W.: The Treatment of Complete Heart Block by the Combined Use of a Myocardial Electrode and an Artificial Pacemaker, Am. Coll. Surgeons, Clin Cong. (43rd), Proc. S. Forum **8**: 360-363, 1958.
19. Cooley, D. A.: Current Status of Surgical Treatment of Ventricular Septal Defect, Dis. Chest **35**: 651-662, 1959.
20. Kay, E. B., Sambhi, M. P., Nogueira, C., and Zimmerman, H. A.: Treatment of Ventricular Septal Defects, J. A. M. A. **165**: 2168-2174, 1957.

(For Discussion, see page 799.)

THE CLOSURE OF ATRIAL SEPTAL DEFECTS UTILIZING GENERAL HYPOTHERMIA

The Effectiveness of Treatment as Determined by Cardiac Catheterization

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In 1954 a number of surgical methods for the correction of atrial septal defect were in use at various centers in this country and in Europe. Among the most popular of these were atrioseptopexy,[1, 2] various other methods of external suture,[3, 4] and the circumclusion operation devised by Sondergaard.[5] The atrial well, introduced by Gross,[6] was found to permit a direct approach to these lesions and this technique was adopted with considerable success by Kirklin and his associates.[7, 8] At about this same time, Lewis[9] and Swan[10] demonstrated the safety and practicability of general hypothermia and showed that the extended period of circulatory occlusion made possible by hypothermia provided opportunity for the suture of atrial septal defects under direct vision. The open operation with hypothermia appeared to have distinct advantages over the various closed or semi-open techniques and this method was selected for use at the National Heart Institute. Between 1954 and 1957, 36 patients with interatrial communications were operated upon with general hypothermia. In 34 patients, in whom repair was carried out, opportunity was afforded for an assessment of the effectiveness of the closure by postoperative cardiac catheterization or autopsy examination. The results of these studies, a description of the techniques employed, and a discussion of the factors limiting the effectiveness of the method are presented.

CLINICAL MATERIAL AND DIAGNOSTIC METHODS

The 36 patients ranged in age from 6 to 49 years; only 11 of them were 15 years or less. With one exception, each presented the characteristic physical, radiographic, and electrocardiographic findings of atrial septal defect. In the 1 patient, subsequently shown to have incomplete atrioventricular canal, the electrocardiogram revealed left axis deviation. Cardiac catheterization was performed in every patient and in each a left-to-right shunt entering the right

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atrium was demonstrated. At the National Heart Institute, the presence, location, and magnitude of left-to-right shunts are determined by the results of indicator-dilution curves and inert foreign gas tests. In the latter method the patient inhales nitrous oxide (N_2O) or radioactive krypton (Kr^{85}) for 30 seconds as blood samples are drawn simultaneously from the right heart or pulmonary artery and a peripheral artery. The presence and magnitude of any left-to-right shunt are indicated by the relative amounts of inert gas present in the two samples.[11, 12] The N_2O test has been proved to be distinctly superior to the method of measuring oxygen differences in several hundred patients studied by both techniques. It not only provides a more sensitive method for detecting a shunt but furnishes a reliable estimate of its magnitude in terms of the ratio of pulmonary to systemic blood flow.[13] A summary of the data obtained at right heart catheterization is presented in Table I.

Significant pulmonary hypertension, that is, a mean pulmonary arterial pressure greater than 22 mm. Hg, was present in 13 patients. In 2 of them (C. R. and T. S.) the pulmonary and systemic pressures were equal and the shunt across the atrial defect was bidirectional. The presence of a sinus venosus type of defect with partial anomalous pulmonary venous drainage was established in 2 patients by the catheter position; indicator-dilution curves were not routinely employed to characterize the drainage pathways of the pulmonary veins at that time.

OPERATIVE TECHNIQUE AND FINDINGS

Every patient was digitalized before operation because of the experimental demonstration that myocardial failure occurring with circulatory occlusion during hypothermia could be prevented by digitalis.[14] For each patient, 500 c.c. of freshly drawn heparinized blood was oxygenated by bubbling oxygen through it and used for gravity perfusion of the coronary arteries during the period of cardiotomy.[15, 16] After the induction of anesthesia with pentobarbital, succinylcholine, and nitrous oxide, the patients were cooled by immersion to an esophageal temperature of 32° to 33° C. The temperature drifted to an ultimate level of 29° to 30° C. at the time of inflow occlusion. Mechanical ventilation with 65 per cent oxygen and 35 per cent nitrous oxide was provided. A right thoracotomy was employed early in the series but median sternotomy was utilized in the majority of patients. After the venae cavae had been encircled with tapes, the ascending aorta was freed to permit subsequent occlusion (Fig. 1). The size and position of the defect, the entrances of the pulmonary veins and the competency of the mitral and tricuspid valves were assessed by palpation through the right atrial appendage (Fig. 2). A partially occluding clamp was applied to the wall of the atrium and a generous incision made within the excluded segment (Fig. 3). The venae cavae were then occluded and after the heart was allowed to empty, the aorta was cross-clamped below the origin of the innominate artery. The coronary perfusion needle (Fig. 3) was inserted into the aorta and oxygenated blood perfused by gravity from a reservoir elevated

TABLE I. HEMODYNAMIC AND OPERATIVE FINDINGS

PATIENT	AGE	PREOP. CATHETERIZATION PA PRESSURE (MM./HG) S/D	MEAN	N_2O% AND LOCATION	TYPE AND SIZE OF DEFECT	POSTOP. CATHETERIZATION PA PRESSURE (MM./HG) S/D	MEAN	N_2O% AND LOCATION	COMMENT
M. L.	36	35/10	21	50 RA†	Secundum, 5 cm.	35/15	25	7 RA	
L. W	24	48/18	32	70 RA†	Secundum, 6 cm.	31/7	17	53 RA	
L. H.	24	30/8	18	——	Secundum, 2 cm.	——	—	——	Died 10th day; autopsy closed
C. R.	35	120/48	74	36 RA	Secundum, 3 cm.	——	—	——	Died 12 hours; autopsy closed
D. McD.	46	40/16	26	85 RA	Secundum, 3 cm.	30/12	19	46 PA	
D. S.	34	80/42	44	83 RA	Sinus venosus	70/28	44	49 PA	
M. K.	22	30/9	16	89 RA	Secundum (fenestrated), 4 cm.	16/6	12	4 PA	
D. B.	41	34/10	21	39 RA	Secundum, 3 cm.	——	—	11 RA	
C. N.	43	74/28	45	43 RA	Secundum, 4 cm.	25/2*	—	29 RA	
E. Del M.	47	85/36	58	48 PA	Secundum, 5 cm.	52/20	38	10 RA	
J. K.	15	23/7	13	67 PA	Secundum, 5 cm.	28/15	18	12 PA	
C. R.	23	28/10	14	66 RA	Secundum, 4 cm.	13/4	7	12 RA	
B. W.	39	32/10	21	48 RA	Secundum, 5 cm.	22/5	9	8 PA	
K. C.	39	42/17	22	72 RA	Secundum, 6 cm.	23/9	15	13 PA	
B. C.	49	34/8	21	32 RA	Secundum, 4 cm.	——	—	——	Died 1 year; no follow-up
B. Q.	18	30/9	16	56 RA	Secundum, 7 cm.	16/5	10	8 PA	
I. T.	37	82/10*	—	48 RA	Secundum, 6 cm.	90/10*	—	42 RV	
C. M.	27	25/11	20	82 RA	Secundum, 6 cm.	22/7	12	40 PA	Reoperated; extracorporeal circulation
J. T.	18	40/15	24	52 RA	Secundum, 7 cm.	——	—	——	No attempt made to repair defect
B. S.	10	35/10*	—	62 RV	Secundum, 5 cm.	18/8	12	8 PA	
D. E.	7	22/5	12	63 RA	Sinus venosus	15/7	10	30 PA	
E. H.	6	35/9	23	56 RA	Secundum, 3 cm.	29/5*	—	13 RA	
M. M.	27	37/7*	—	61 RV	Atrioventricular canal	——	—	——	Died at operation
W. H.	18	25/6	17	71 RA	Sinus venosus	22/8	14	36 PA	
R. J.	9	15/4	7	44 RA	Secundum, 3 cm.	26/9	15	23 PA	
B. S.	14	25/8	16	62 PA	Secundum, 3 cm.	19/8	12	7 PA	
S. H.	14	24/7	12	57 RV	Secundum, 2 cm.	23/10	14	12 PA	
C. O.	10	18/6	12	50 PA	Secundum, 4 cm.	22/7	12	12 PA	
L. B.	27	22/10	12	54 PA	Secundum (fenestrated), 5 cm.	18/6	9	11 PA	
M. E.	32	35/10	16	70 PA	Secundum, 3 cm.	25/10	13	12 PA	
M. K.	9	26/10	16	59 PA	Sinus venosus	16/2	6	8 RA	
R. R.	9	28/15	21	37 PA	Secundum, 3 cm.	32/15	23	6 PA	
L. W.	47	34/15	19	60 PA	Secundum (fenestrated), 7 cm.	25/6	14	8 PA	
G. R.	37	24/14	16	32 PA	Secundum, 3 cm.	18/7	12	5 PA	
D. S.	12	125/70	95	38 PA	Secundum, 2 cm. Reverse ductus	120/45	70	0 RA 10 PA	Died following closure reverse ductus
C. A.	31	20/7	12	51 PA	Secundum, 3 cm.	27/13	18	73 PA	Reoperated; extracorporeal circulation

*Right ventricular pressure.
†Nitrous oxide index calculated from oxygen differences.

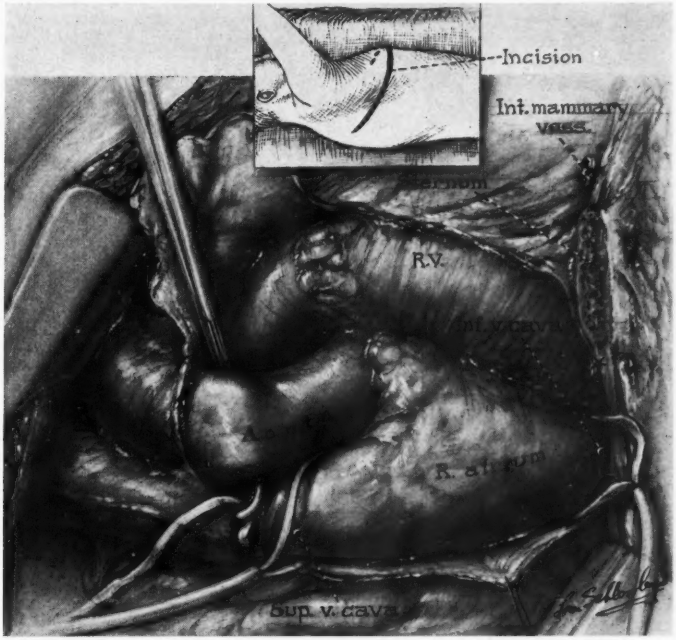

Fig. 1.—Surgical exposure employed in the direct closure of atrial septal defects with general hypothermia. A median sternotomy rather than a transverse thoracotomy was most often employed.

Fig. 2.—The location and size of the defect and the consistency of its margins are assessed by digital examination before circulatory occlusion.

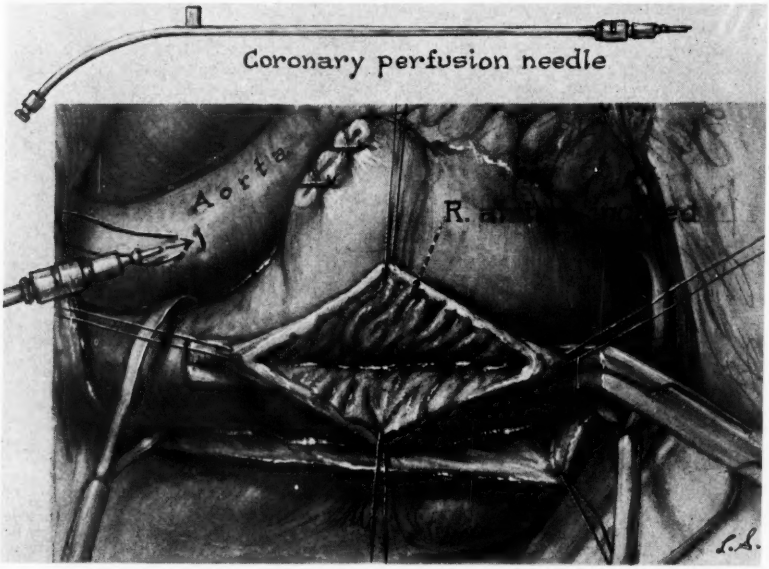

Fig. 3.—The right atriotomy is made over a partially occluding clamp. The coronary perfusion needle (designed by Dr. James Jude) will be inserted through the mattress suture in the wall of the aorta.

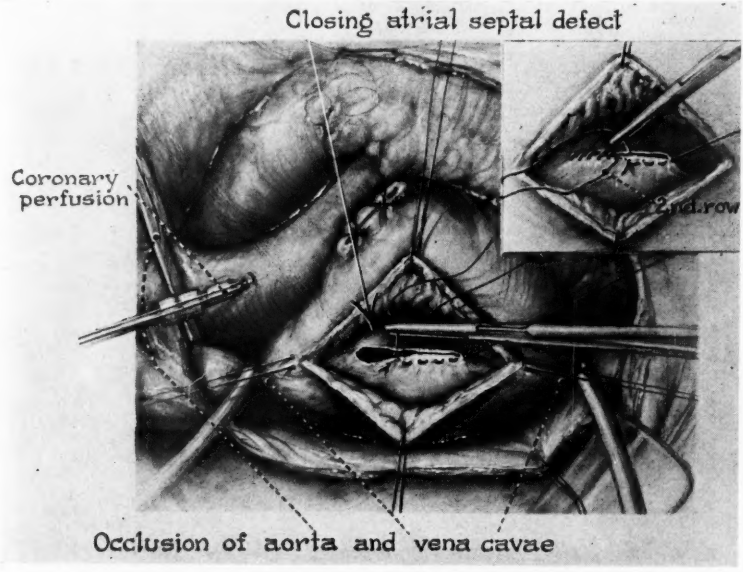

Fig. 4.—The defect is closed by two layers of sutures as shown

100 cm. above the table. In the first 9 patients, the defect was closed with a single continuous suture, but, in all subsequent ones, two layers of sutures were placed. The first was a continuous everting mattress suture which was drawn up after it had been placed and as the left atrium was flooded with blood from the inferior vena cava. The everted margins of the defect were then approximated by a second over and over row (Fig. 4). The right atrium and ventricle

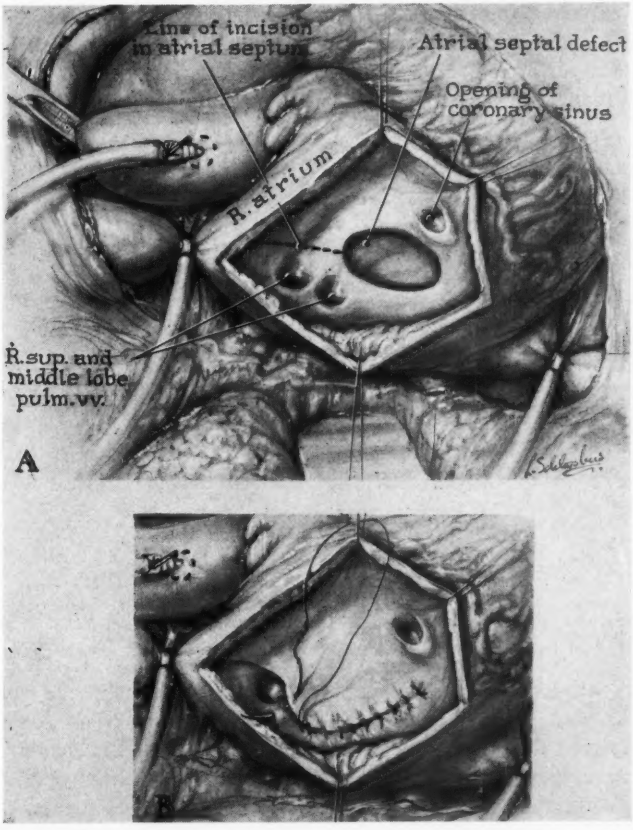

Fig. 5.—The method employed for the correction of the sinus venosus type of defect by direct suture. (From Bahnson, Spencer, and Neill: J. THORACIC SURG. 36: 777, 1958.)

were then filled with blood, the clamp reapplied to the atriotomy and venous return restored. The periods of circulatory occlusion ranged from 3 to 9 minutes and averaged 5.5 minutes.

In 2 of the 4 patients with sinus venosus defects and anomalous drainage of the upper and middle lobe pulmonary veins, an attempt at complete correction was made by the method described by Bahnson[17] (Fig. 5). In the others, the atrial defect was closed and the pulmonary arteries supplying the upper and middle lobes were ligated. A description of the lesion encountered in the remaining patients is included in Table I.

RESULTS

Three patients died after operation. In 1 (M. M.) the unsuccessful attempt at correction of incomplete atrioventricular canal was followed by irreversible ventricular fibrillation. Patient C. R., with pulmonary hypertension and bidirectional shunt, died after 12 hours, and L. H. died suddenly on the tenth postoperative day after the onset of an acute arrhythmia. In each of these 2 patients, complete closure of the defects was demonstrated at autopsy. A fourth patient (B. C.) died at home 1 year after operation; no postoperative catheterization had been performed. In 1 patient (J. T.), a defect, estimated by palpation to be more than 7 cm. in diameter, was present and closure was not attempted.

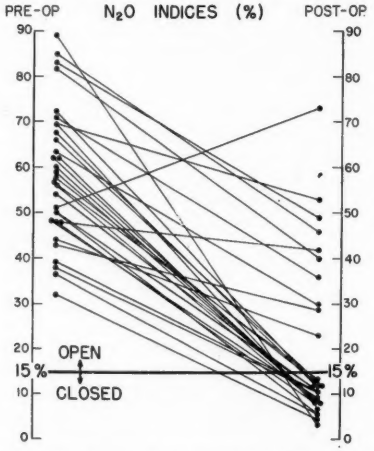

Fig. 6.—Nitrous oxide indices recorded at cardiac catheterization before *(left)* and after *(right)* operation in 31 patients with septum secundum or sinus venosus defects. A nitrous oxide index of 15 per cent or greater indicates the presence of a left-to-right shunt. Indices of 40 and 60 per cent indicate pulmonary to systemic flow ratios of 1.5 and 2.5, respectively.

Postoperative cardiac catheterizations were carried out in the 31 surviving patients with sinus venosus or septum secundum defects in whom correction had been attempted. The results of these studies are presented in detail in Table I and shown graphically in Fig. 6. The N_2O indices were clearly indicative of a residual left-to-right shunt in 10 patients. In 7 of these patients, the shunts were of relatively large magnitude and the pulmonary to systemic flow ratios were 1.4:1 or greater. In each patient in whom the results of N_2O tests indicated a residual shunt, arterial indicator-dilution curves recorded after right heart injection were also abnormal and confirmed the presence of a persistent communication.

COMMENT

Atrial septal defects of the usual or secundum variety are anatomically distinct from those of the sinus venosus type and the results of operative treatment must be considered accordingly. In each of the patients with septum

secundum defects, regardless of size, closure was of necessity carried out by direct suture and it was considered likely that the frequency of incomplete repair would be higher among patients with defects of large size. In Fig. 7 the incidence of complete repair is plotted in relation to the size of the defect encountered at operation. It can be seen that there is no absolute correlation between the size of the defect and the incidence of incomplete closure. It was

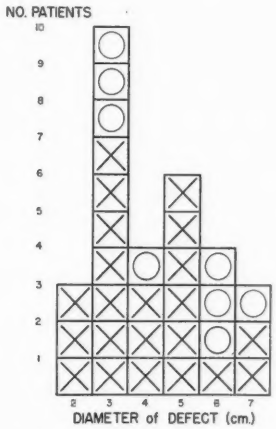

Fig. 7.—Effectiveness of the repair of septum secundum defects in relation to their size. Complete closure is indicated by a *cross*, a residual communication by a *circle*.

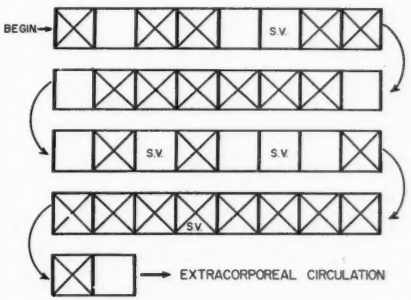

Fig. 8.—Effectiveness of repair in relation to the chronologic order in which the patients were operated upon. The initials *S.V.* indicate sinus venosus defects.

of interest, however, that of the seven largest defects (6 cm. or greater in diameter) four were open and of the thirteen smallest ones (3 cm. or less in diameter) only three were incompletely repaired. In Fig. 8 the operative result is similarly indicated but the patients are arranged in the chronological order in which the operations were carried out. It is apparent that complete closure was achieved with greater frequency late in the series, reflecting increased familiarity with the operation and more deliberate attention to the repair. It should be pointed out, however, that among the first 16 patients only 1 was a child, aged 15 years

or less, while among the last 18 patients 10 were in this age group. When age alone was considered in relation to the effectiveness of the operative treatment of secundum defects it was found that among the 9 children only 1 had a residual shunt while in the 21 adults a residual shunt was present in 7. There was no correlation between the adequacy of repair and the duration of circulatory occlusion.

In 2 patients with sinus venosus defects (D. S. and D. E.) only the interatrial communication was closed and the segmental pulmonary arteries supplying the upper and middle lobes were ligated. In each, the magnitude of the left-to-right shunt was reduced, but the residual shunt, presumably from the anomalous veins, was surprisingly large. In 2 additional patients (W. N. and M. K.), complete correction of the malformation by direct suture was attempted (Fig. 5) and was successful in 1 of them. A disadvantage of direct suture in the correction of sinus venosus defects is danger of constriction of the superior vena cava and subsequent thrombosis of it or one of the pulmonary veins. This complication was not observed, however, in either patient.

No detailed correlation was made between the clinical and hemodynamic findings at the time of postoperative study. In general, however, the presistence of fixed splitting of the pulmonary second sound was indicative of a large residual shunt. In some patients, however (for example, R. J.), normal closure of the second sound during expiration was noted postoperatively and, on the basis of the physical findings alone, the defect would have been considered completely closed.

In the latter part of the operative series, arterial indicator-dilution curves were recorded, after injection into the right superior pulmonary vein, before the thoracotomy was closed. Curves of normal contour were obtained at operation in 2 patients (C. M. and R. J.) who were later shown at catheterization to have residual shunts. It seems probable, therefore, that complete closure was initially achieved in these patients but that the suture line dehisced in the postoperative period. This view is substantiated by the findings in 2 patients with residual shunts of large magnitude who have been operated upon again with the aid of extracorporeal circulation. In each, a series of holes of varying size was present in the initial suture line. It was apparent that many sutures had torn through the septum and the initial single defect had only been converted into many smaller ones.

The primary cause for incomplete correction among the patients described is considered to be the relatively brief period of cardiotomy provided by general hypothermia. It was often difficult to define precisely the margins of the defect and direct suture was always necessary, even when it resulted in a closure under tension. In many patients there was obvious need for prosthetic replacement of a portion of the septum but the insertion of such material was patently impossible in the time allowed. Although repetitive periods of occlusion have been employed by Kirby[18] and others to extend the total period of cardiotomy, even this technique would not seem adequate for the time-consuming reconstruction

often necessary. For these reasons, the use of general hypothermia in the treatment of patients with atrial septal defect has been abandoned and the correction of this malformation is always undertaken during the prolonged period of circulatory exclusion made possible by extracorporeal circulation. The advantages of the latter method seem apparent at this time but proof of its value will, of course, depend upon a detailed hemodynamic evaluation such as that presented above.

SUMMARY

In 34 patients with atrial septal defects of the septum secundum or sinus venosus type, correction was attempted by direct suture during a period of circulatory occlusion provided by general hypothermia. The effectiveness of the repair was assessed in 33 by postoperative cardiac catheterization or autopsy examination. A residual left-to-right shunt was present in 7 of 29 patients with secundum defects and in 3 of 4 patients with sinus venosus defects. These poor results were attributed to the brief period of cardiotomy provided by hypothermia which necessitated closure by direct suture in every instance. Since extracorporeal circulation permits a prolonged intracardiac procedure and the insertion of a prosthesis if necessary, this technique is now considered preferable in the treatment of all patients with atrial septal defect.

REFERENCES

1. Bailey, C. P., Downing, D. F., Geckeler, G. D., Litkoff, W., Goldberg, H., Scott, J. C., Janton, O., and Redondo-Ramirez, H. P.: Congenital Interatrial Communications: Clinical and Surgical Considerations With a Description of a New Surgical Technic: Atrio-Septo-Pexy, Ann. Intern. Med. **37**: 888, 1952.
2. Bailey, C. P., Bolton, H. E., Jamison, W. L., and Neptune, W. B.: Atrio-Septo-Pexy for Interatrial Septal Defects, J. THORACIC SURG. **26**: 184, 1953.
3. Watkins, E., Jr., and Gross, R. E.: Experiences With Surgical Repair of Atrial Septal Defects, J. THORACIC SURG. **30**: 469, 1955.
4. Bahnson, H. T.: Accomplishments and Possibilities in Cardiovascular Surgery, Am. J. Roentgenol. **76**: 730, 1956.
5. Sondergaard, T.: Closure of Atrial Septal Defects. Report of Three Cases, Acta chir. Scandinav. **107**: 492, 1954.
6. Gross, R. E., Watkins, E., Pomeranz, A. A., and Goldsmith, E. I.: A Method for Surgical Closure of Interauricular Septal Defects, Surg. Gynec. & Obst. **96**: 1, 1953.
7. Kirklin, J. E., Swan, H. J. C., Wood, E. H., Burchell, H. B., and Edwards, J. E.: Anatomic, Physiologic, and Surgical Considerations in Repair of Interatrial Communications in Man, J. THORACIC SURG. **29**: 37, 1955.
8. McGoon, D. C., Swan, H. J. C., Brandenburg, R. O., Connolly, D. C., and Kirklin, J. W.: Atrial Septal Defect: Factors Affecting the Surgical Mortality Rate, Circulation **19**: 195, 1959.
9. Lewis, F. J., and Taufic, M.: Closure of Atrial Septal Defects With the Aid of Hypothermia; Experimental Accomplishments and the Report of One Successful Case, Surgery **33**: 52, 1953.
10. Swan, H.: Surgical Closure of Interauricular Septal Defects, J. A. M. A. **151**: 792, 1953.
11. Morrow, A. G., Sanders, R. J., and Braunwald, E.: The Nitrous Oxide Test: An Improved Method for the Detection of Left-to-Right Shunts, Circulation **17**: 284, 1958.
12. Sanders, R. J., and Morrow, A. G.: The Diagnosis of Circulatory Shunts by the Nitrous Oxide Test. Improvements in Technic and Methods for Quantification of the Shunt, Circulation **18**: 856, 1958.
13. Sanders, R. J., Cooper, T., and Morrow, A. G.: An Evaluation of the Nitrous Oxide Method for the Quantification of Left-to-Right Shunts: An Experimental Comparison of the Gasometric Technique With Directly Metered Blood Flows, Circulation **19**: 898, 1959.

14. Lombardo, T. A., Radigan, L. R., and Morrow, A. G.: Myocardial Failure in Experimental Hypothermia, Circulation Res. 5: 22, 1957.
15. Shumway, N. E., Gliedman, M. L., and Lewis, F. J.: Coronary Perfusion for Larger Periods of Cardiac Occlusion Under Hypothermia, J. THORACIC SURG. 30: 598, 1955.
16. Jude, J. R., Fauteux, J. P., and Harontunian, L. M.: Differential Cardiac Warming During Hypothermia, S. Forum 7: 294, 1956.
17. Bahnson, H. T., Spencer, F. C., and Neill, C. A.: Surgical Treatment of Thirty-Five Cases of Drainage of Pulmonary Veins to the Right Side of the Heart, J. THORACIC SURG. 36: 777, 1958.
18. Johnson, J., Kirby, C. K., Blakemore, W. S., and Zinsser, H. F.: Intermittent Inflow Occlusion for the Direct Visual Repair of Atrial Septal Defects, J. THORACIC SURG. 37: 314, 1959.

(For Discussion, see page 799.)

OPEN-HEART SURGERY USING DEEP HYPOTHERMIA WITHOUT AN OXYGENATOR

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THE DEVELOPMENT of efficient heat exchangers for extracorporeal cooling of blood has now made possible prolonged intracardiac surgical procedures using deep hypothermia without a mechanical oxygenator. Detailed physiologic studies during deep hypothermia have been performed on dogs[1] and we have used it for the surgical repair of a variety of cardiac malformations. This paper reports our first 27 consecutive patients with congenital cardiac defects operated upon by this technique.

REQUIREMENTS FOR DEEP HYPOTHERMIA

Moderate hypothermia by external cooling is usually limited to temperatures of 26° to 28° C. because of the danger of cardiac arrhythmias and ventricular fibrillation below this level. There are two requirements for further cooling; (1) an efficient system for rapid cooling and rewarming of the body; and (2) a dual pump system to support the pulmonary and systemic circuits when cardiac arrest or ventricular fibrillation occur at deep hypothermic levels.

The basic equipment and hook-up for deep hypothermia is diagrammed in Fig. 1. This is essentially the same plan used by Drew[2, 3] and Kenyon,[4] in England, and Shields and Lewis[5] in this country. Systemic venous blood drains by gravity from the right atrium into a reservoir from which it is pumped back into the pulmonary circuit. Arterialized blood drains by gravity from the left atrium into a reservoir from which it is pumped through a heat exchanger and filter into the iliac artery. Occlusive roller pumps have been used and the heat exchanger is the Brown-Harrison model.[6] To produce cooling, the heat exchanger has been used in conjunction with a recirculation bucket containing crushed ice and water which maintains a temperature of between 0° and 2° C. For rewarming, hot and cold tap water are used and maintained at a temperature of 42° C. by the heat exchanger regulator. A single heat exchanger is used for patients weighing up to 80 pounds, a double heat exchanger for patients over this amount.

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OPERATIVE DETAILS

Metal-tipped plastic catheters are used for cannulation of the right and left atrial appendages. The left atrium is more difficult to cannulate than the right, especially if it is small or there is clockwise rotation of the heart. A plastic catheter is then inserted and secured in the pulmonary artery and a metal cannula in the iliac artery. The catheter in the pulmonary artery is usually introduced through a stab wound in an avascular portion of the right ventricular outflow tract, just below the pulmonary valve. It is threaded up through the valve and secured in place by means of a purse-string suture at the point of insertion. In the presence of severe pulmonary stenosis this catheter is inserted and secured in a similar fashion in the proximal portion of the main pulmonary artery.

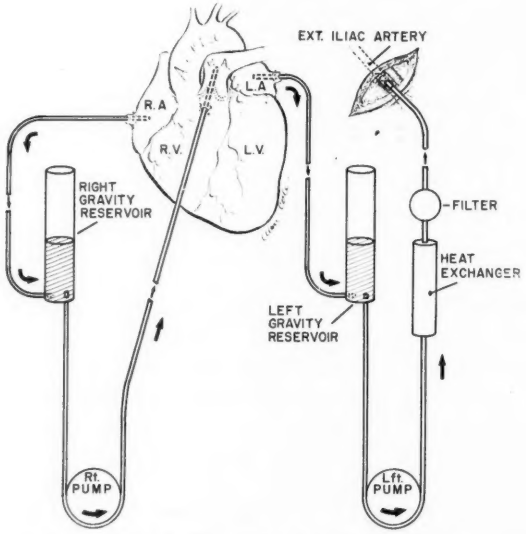

Fig. 1.—Circuit and equipment for deep hypothermia.

During cooling, the anesthetic agent is administered in a mixture of 95 per cent oxygen and 5 per cent carbon dioxide. When the temperature reaches 28° C., the anesthetic agent is discontinued but pulmonary ventilation is maintained.

The gravity reservoirs are adjusted for maximum drainage and the left, or systemic, circuit is started first. As soon as it is stabilized, the right, or pulmonary, circuit is activated. As the body temperature falls, the arteriovenous oxygen difference is reduced as a result of the marked decrease in oxygen consumption. The arteriovenous oxygen difference becomes negligible or absent at temperatures between 10° and 14° C.

When the body temperature has been lowered to the desired level, the pumps are turned off and the heart is isolated as illustrated in Fig. 2. The

aorta (*1*) is cross-clamped for two reasons: (a) to prevent the escape of any air which might reach the left side of the heart through an atrial or ventricular defect when the right side is opened; and (b) to prevent blood from flowing back through the coronary arteries into the right heart where it would obscure the field and require aspiration. Previously placed tapes are pulled up and secured on the superior (*2*) and inferior (*3*) venae cavae.

The heart is then drained into the gravity reservoirs via the atrial catheters. The right atrial (*4*) and left atrial (*5*) lines are then clamped. The clamps on the atrial lines are not essential, but this is a useful maneuver since it prevents them from filling with air during the time the heart is open and bloodless.

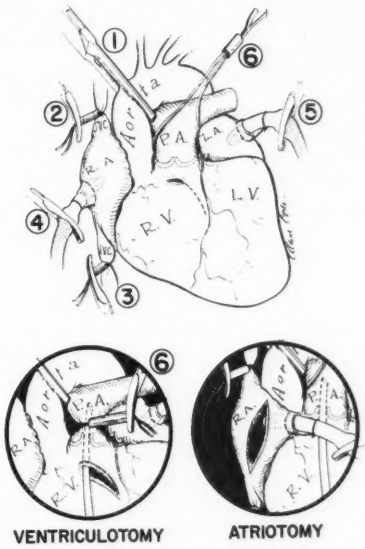

Fig. 2.—Operative details for deep hypothermia.

Above: Pulmonary artery catheter removed in preparation for right ventriculotomy. *1*, Aortic clamp. *2*, Superior vena cava pull-up tape and clamp. *3*, Inferior vena cava pull-up tape and clamp. *4*, Right atrial catheter clamp. *5*, Left atrial catheter clamp. *6*, Pulmonary artery pull-up tape and clamp.

Lower left: Pulmonary artery catheter reinserted through the ventriculotomy and tape around pulmonary artery pulled up for filling left side of heart with blood from pulmonary pump.

Lower right: Position of catheters and appendage for right atriotomy.

When a ventriculotomy is performed, the catheter in the pulmonary artery is removed and the stab wound is extended for exposure of the ventricular septum. During the surgical procedure the heart remains bloodless and is completely quiet as a result of cardiac arrest or fine ventricular fibrillation. Before the final sutures of a ventricular repair are tied, the pulmonary artery catheter is reinserted through the ventriculotomy, as shown in the lower left offset of Fig. 2. The previously placed tape around the pulmonary artery (*6*) is pulled up and secured. The pulmonary circuit pump is then turned manually. This fills the left atrium and ventricle with blood, which spills over into

the right ventricle. When all air has been expelled, the final sutures in the ventricular septum are tied down under blood. The pulmonary circuit is again pumped manually to test for leaks in the closure as well as for fenestrations or additional defects in the septum. When satisfactory closure has been achieved, the tape on the pulmonary artery (*6*) is released and the ventriculotomy is closed, leaving the pulmonary artery catheter in one end of the incision. Before this closure is completed, the caval tapes (*2* and *3*) are released to allow the right heart to fill with blood. The clamps (*4* and *5*) on the atrial lines are then removed and any residual air in these lines or the heart is displaced into the gravity reservoirs.

The pumps are then turned on and rewarming is started. The aortic clamp (*1*) is left in place for a minute or two during which the heart is manipulated to displace any residual air into the right or left atrial lines. The clamp on the aorta is then removed as rewarming continues.

For lesions involving the atrium, essentially the same technique is applied, except that it is unnecessary to remove the pulmonary artery catheter. The right atrial catheter is displaced to the left as shown in the lower right offset of Fig. 2 and atriotomy is performed without removal of the atrial line. The pulmonary circuit is pumped manually to exclude all air from the left side of the heart before the final atrial septal sutures are secured. Closure and rewarming are then performed in a fashion similar to the above.

For aortic or pulmonary valvular lesions, the technique is essentially the same. The pulmonary artery catheter is removed during repair of the pulmonary valve, whereas, all catheters are left in place for aortic valvular surgery.

DURATION OF CIRCULATORY ARREST

Since this technique involves complete arrest of the circulation, it is essential to know the duration of circulatory arrest which can be tolerated at various temperatures without damage to the brain or myocardium.

Studies of oxygen consumption at various temperatures by Bigelow,[7] Gollan,[8] and others, have helped us in this regard. However, their studies did not include the temperature ranges in which we are interested for prolonged deep hypothermia. Therefore, we have studied the oxygen consumption in a group of 10 dogs in which the body temperature was reduced to 6° C. For these studies, the temperature and circulation were maintained at the various levels by means of extracorporeal pumps and a heat exchanger.

The results of these studies are summarized in Fig. 3. This is a simplified break-down of the data in which each point on the graph represents a 50 per cent reduction of oxygen consumption from the previous point. At 29° C., oxygen consumption is 50 per cent of normal. It is further reduced to 25 per cent at 22° C., to 12 per cent at 16° C., to 6 per cent at 10° C. and to 3 per cent at 6° C. Using the hypothesis that each 50 per cent reduction in oxygen consumption doubles the safe period for circulatory arrest, we can construct the table shown in Fig. 3. To corroborate this hypothesis, dogs have been cooled as low as 10° C. and subjected to 60 minutes of total circulatory arrest. After

rewarming they made uneventful recoveries and resumed normal activities without evidence of central nervous system damage or personality alteration. These studies are reported in detail elsewhere.

These results have now been further corroborated by our clinical cases in which patients at similar temperatures have had intracardiac surgery with complete recovery to normal following circulatory arrest in excess of 1 hour.

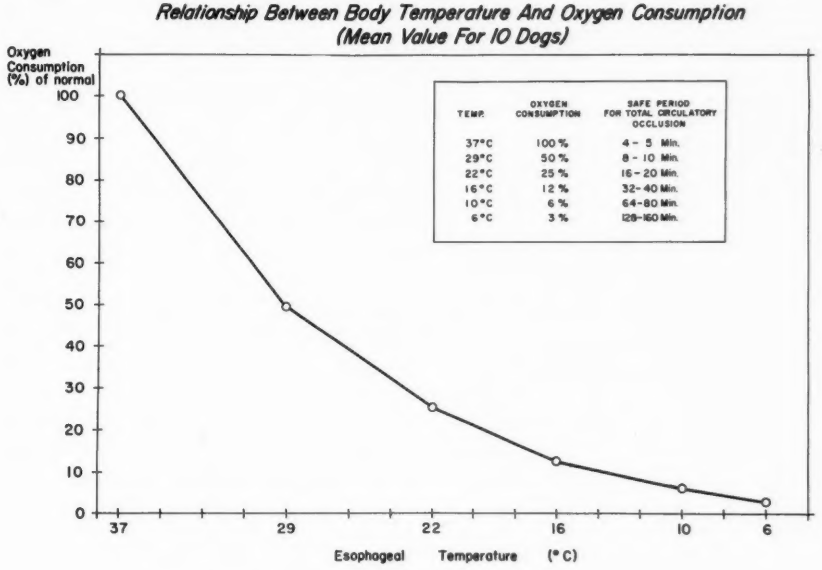

Fig. 3.

CLINICAL EXPERIENCES

This technique of deep hypothermia was first used by us clinically in August, 1959, for the repair of a supravalvular aortic stenosis in an 8-year-old boy. The stenosis was caused by a constriction of the aorta similar to a coarctation located 0.5 cm. distal to the aortic annulus. It was successfully repaired without creating any regurgitation by the insertion of a teflon patch in the coarcted area during 55 minutes of circulatory arrest with the body temperature at 13° C. He made an uneventful recovery and 1 year later was doing well and engaging in full normal activities.

Twenty-six additional patients with congenital cardiac defects have been operated upon under deep hypothermia since that time and are summarized in Table I. The variety of lesions in this group indicates that this modality is applicable to most congenital cardiac lesions. Only one patient with aortic valvular stenosis died and this resulted not from the use of deep hypothermia but rather from a technical error resulting in complete closure of the right coronary artery orifice in the repair of the aortotomy. The two deaths in the

group having ventricular septal defects resulted from aspiration of food by one child on the third postoperative day and a complete atrioventricular block caused death on the eighteenth postoperative day in the other.

TABLE I. TWENTY-SEVEN CONSECUTIVE PATIENTS WITH CONGENITAL CARDIAC DEFECTS OPERATED UPON UNDER DEEP HYPOTHERMIA

DIAGNOSIS	NUMBER OF CASES		
	PERFORMED	RECOVERED	DIED
Atrial septal defect	3	3	
Total anomalous pulmonary veins with ASD	2	2	
Aortic stenosis			
Valvular	3	2	1*
Supravalvular	1	1	
Pulmonary stenosis			
Valvular and infundibular	2	2	
Valvular and atrial septal defect	1	1	
Valvular, infundibular, and atrial septal defect	2	2	
Ventricular septal defect	6	4	2†
Tetralogy of Fallot	7	3	4‡
Totals	27	20	7

*Technical error—Right coronary artery totally obstructed in closure of aorta.

†One VSD aspirated food on third postoperative day; other 1 died on eighteenth postoperative day with complete block.

‡One patient with tetralogy of Fallot had common ventricular chamber; 2 had absent pulmonary valve; 1 had associated ASD.

The higher mortality in the cases of tetralogy of Fallot has not been completely explained. The deaths were all in patients who had defects in addition to the usual tetralogy. One had an endocardial cushion-type of defect; 2 had congenital absence of the pulmonary valve; and 1 had an associated atrial septal defect.

The over-all mortality in this entire group of unselected cases is 26 per cent. Excluding the cases of tetralogy, the mortality is 15 per cent.

Table II summarizes the characteristics of these 27 patients and their perfusion under deep hypothermia. The lowest body temperature used was 9° C. for a patient with aortic stenosis who had 63 minutes of circulatory arrest. Most of the patients were cooled to between 10° and 14° C. The temperatures of the patients with atrial septal defects were lowered to only 20° or 21° C.

TABLE II. TWENTY-SEVEN CONSECUTIVE PATIENTS WITH CONGENITAL CARDIAC DEFECTS OPERATED UPON UNDER DEEP HYPOTHERMIA

Age	3½-26 years
Weight	29-160 pounds
Lowest temperature	9°-21° C.
Cooling rate	1.5° C. per minute
Circulatory arrest time	18-63 minutes
Temperature drift upward during circulatory arrest	
15 minutes	1°-2° C.
30 minutes	3°-4° C.
60 minutes	5°-6° C.
Rewarming rate	1° C. per minute
Blood flow rate	60-100 c.c./Kg./min.
Incidence of ventricular fibrillation	All except 3 cases

Extracorporeal cooling of blood results in more rapid and greater cooling of the "core" organs, such as the heart, brain, liver, etc., than of the great

muscle and skin masses of the body. In this way it differs from external cooling in which skin and muscle are cooled first and central organs secondarily. It also differs from external cooling in that when the pumps are turned off and cooling is discontinued, the central temperature drifts upward instead of downward because the muscle mass is warmer than the core organs. This phenomenon is illustrated in Table II where it is noted that the temperature drift upward during circulatory arrest usually amounts to 1° to 2° C. in 15 minutes; 3° to 4° C. in 30 minutes; and 5° to 6° C. in 1 hour. This factor must be taken into consideration in calculating the anticipated duration of circulatory arrest. The mean temperature during this period should be used.

The flow rate depends upon the amount of gravity used and the size of the atrial catheters. Using 14 to 18 inches of gravity and either 5/16 or 3/8 inch catheters, the flow rates varied between 60 and 100 c.c. per kilogram per minute. These were adequate to cool the patients at a mean rate of 1.5° C. per minute. Rewarming always takes slightly longer. This would mean that cooling a patient from 37° C. to 10° C. would require 18 minutes and rewarming about 22 minutes.

As the temperature decreases, the blood flow rate also decreases as a result of increasing viscosity. However, even at the lowest temperatures, we were usually able to maintain mean blood pressures of 50 mm. Hg or over. During rewarming, the initial blood flow rate starts slowly and gradually increases as the temperature rises.

OTHER PHYSIOLOGIC DETERMINATIONS

Temperature Response.—Studies on dogs, which will be reported in detail elsewhere, have indicated the value of using esophageal temperature in these cases. We have measured skin, muscle, rectal, esophageal, brain, superior vena cava, and right gravity reservoir temperatures during cooling, circulatory arrest, and rewarming. Some of these results are shown graphically in Fig. 4. It will be seen that the esophageal and brain temperatures drop rapidly during cooling, drift up almost the same amount during arrest, and rise rapidly during rewarming. In these studies, the esophagus was usually about 1° C. warmer than the brain, although it was occasionally a degree cooler. This means that accurate monitoring of the esophageal temperature provides an index of brain temperature, plus or minus one degree. Rectal temperatures, even after cleansing enemas, failed to parallel esophageal temperatures closely. The lag in muscle temperature change is also shown in the diagram, and its influence on the upward drift of brain and esophageal temperature during circulatory arrest is obvious.

The esophageal temperature usually drifted upward more than the brain temperature. This is probably an anatomic effect, since the core organs in the trunk are surrounded by a generous muscle and subcutaneous mass, whereas, the brain is more isolated and the drift of the temperature tends to be less.

At the completion of rewarming to temperatures of 36° to 37° C. the esophageal temperature always drifts downward several degrees since the muscle mass remains cooler than the core organs.

Electrocardiographic Responses.—Ventricular fibrillation occurred in all except 3 of the patients. In these latter cases, a normal rhythm resumed spontaneously. Ventricular fibrillation usually occurred below 18° C., although occasionally at higher temperatures. Those hearts which did not fibrillate during cooling, usually went into ventricular fibrillation during the surgical procedure. However, the cooler the temperature, the finer is the fibrillation. During cardiotomy, if ventricular fibrillation is present it is so fine that there is no gross

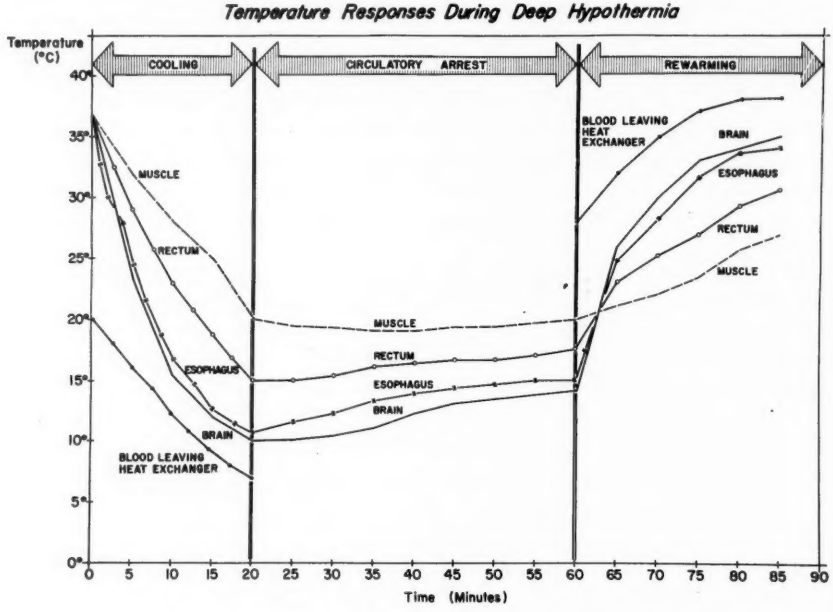

Fig. 4.

movement and it can only be detected on the electrocardiogram. Most of the hearts followed a characteristic electrocardiographic pattern which included early premature ventricular contractions, followed by atrial fibrillation and progressing into bradycardia, coarse ventricular fibrillation and, ultimately, fine ventricular fibrillation (Fig. 5). During the period of circulatory arrest, the heart frequently changed back and forth between ventricular fibrillation and cardiac arrest, with an occasional electrocardiographic complex.

Rewarming usually initiated a fine fibrillation which gradually became more coarse. The hearts were defibrillated when the temperature reached 30° C. Most of them were defibrillated with a single shock. The heart with the right coronary artery sutured closed required three shocks, and one other heart required two shocks.

Electroencephalographic Response.—All of the patients had preoperative and postoperative complete electroencephalograms. All of these were read as

normal. There was no evidence of any neurological, central nervous system, personality, or behavior alteration in any of these cases.

Electroencephalograms taken during the procedures usually followed a characteristic pattern (Fig. 6). During the early stages of cooling, alpha activity increased perceptibly but, later, slowed and large delta waves appeared. Below 16° to 18° C., activity almost ceased, although some very fine waves persisted. However, these probably represent artifacts, such as the activity of the pumps or electrical potential in the skin, rather than brain waves. The electroencephalogram remained completely flat during circulatory arrest. It

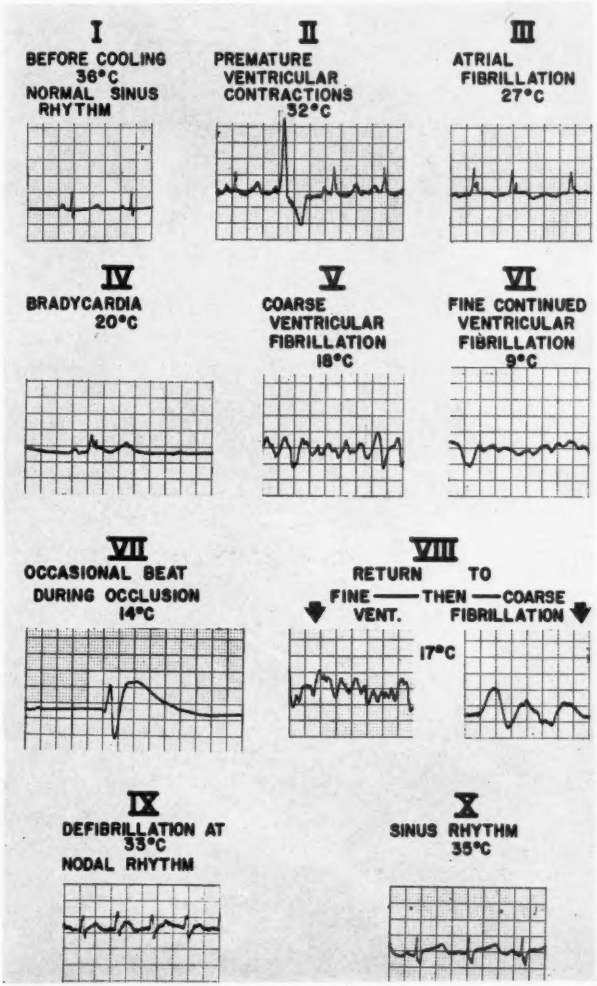

Fig. 5.—Electrocardiographic response to deep hypothermia.

was re-activated during rewarming, but the patterns returned much more slowly than they had disappeared during cooling. In fact, the electroencephalogram lagged far behind the brain temperature in returning to normal. This was a common finding for which we have no explanation. However, the electroencephalogram was back to normal within 20 to 40 minutes after rewarming had been completed.

Metabolic Effects.—The metabolic responses during and after 2 typical cases of deep hypothermia are summarized in Table III. There were no significant changes in arterial or venous pH, CO_2 content, lactic acid, or pyruvic acid during cooling to 10° C. All pH samples were run at the same temperature

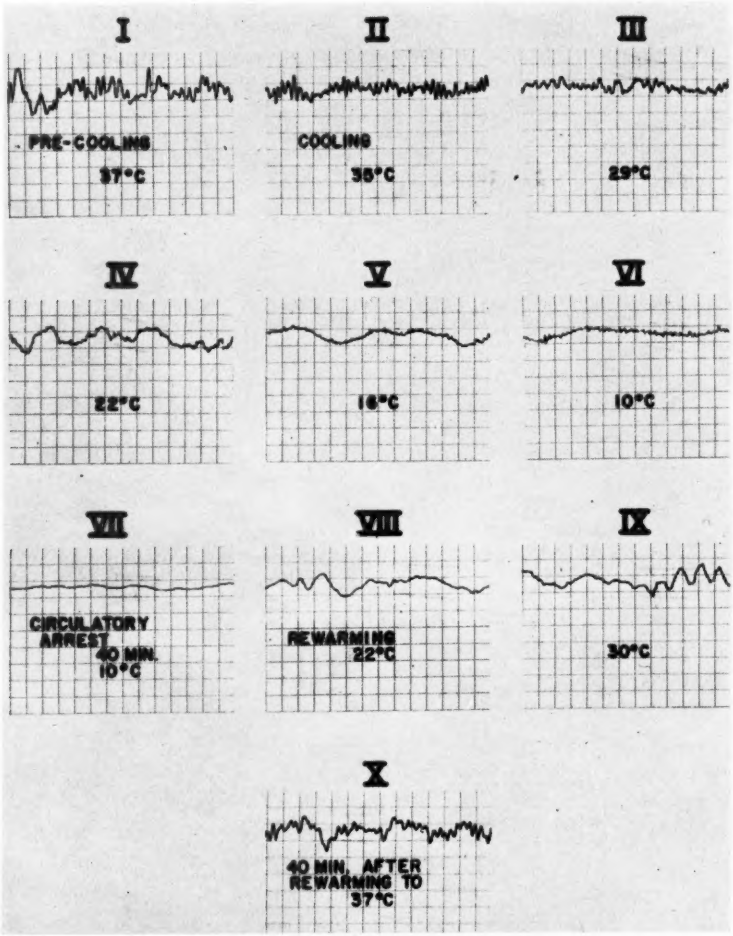

Fig. 6.—Electroencephalographic pattern during deep hypothermia.

at which they were drawn. This is essential since temperature change affects hydrogen ion dissociation and acidotic readings will be obtained if determinations are made at standard temperature.

TABLE III. METABOLIC EFFECTS OF DEEP HYPOTHERMIA, CASES 24 AND 25 (MEAN VALUES)

TIME OF DETERMINATION	pH		CO_2 CONTENT (MEQ./L.)	LACTIC ACID (MG. %)	PYRUVIC ACID (MG. %)
	ARTERIAL	VENOUS			
Before cooling	7.49	7.42	16	14.7	0.57
During cooling					
22° C.	7.46	7.42	18		
16° C.	7.46	7.42	17		
10° C.	7.44	7.40	19	20.1	0.54
Start rewarming	7.24	7.22	12	28.8	0.57
Finish rewarming	7.29	7.23	11	35.0	0.82
Postoperative					
2 hr.		7.28	14	32.8	0.41
8 hr.		7.32	19	14.5	0.64
24 hr.		7.34	22	16.3	0.54
48 hr.		7.36	23	17.3	0.68

Diagnosis: Ventricular septal defect.
Circulatory arrest: 40 minutes.
Lowest temperature: 10° C.

During rewarming the pH became somewhat acidotic. This resulted from the flushing out of acid metabolites which had accumulated in the tissues during circulatory arrest. The drop in buffer base and the rise in lactic acid during rewarming are responsible for the pH change.

Within 2 hours after surgery, all metabolic determinations were returning to normal and, by 8 hours, they had reached normal ranges, where they remained. The pyruvic acid rose somewhat during the rewarming phase, but it remained within normal limits throughout the procedure.

Postoperatively these patients did not appear acidotic and none of them required any alkaline medication.

ADVANTAGES OF DEEP HYPOTHERMIA

During the past several years it has become increasingly evident that successful intracardiac surgery can be performed using various techniques and various types of equipment. Some groups have even found it advantageous to combine several methods, such as deep hypothermia with a pump-oxygenator.[9-12] It would appear that results are related not to the specific technique or equipment used but, rather, to the background and experience of the pump team, to the type and severity of cases selected for open-heart surgery, and to the skill and experience of the surgeon.

Accordingly, a change to the use of deep hypothermia should be based upon distinct advantages that it offers over other methods for open-heart surgery. We believe its advantages are threefold: (1) mechanical simplicity, (2) reduced blood volume, and (3) an ideal operative field.

The mechanical simplicity speaks for itself. The elimination of the oxygenator with its adjuncts and monitoring devices not only has simplified the

system, but has completely eliminated such problems as siliconizing, foaming, level control, flow control, and hemolysis. The use of hypothermia has made it possible to stop the pumps at any time during the cooling or rewarming for whatever eventuality may arise.

The total priming volume for the complete system is 1,200 c.c. of heparinized blood. This volume is the same for all patients, except for the addition of 150 c.c. when a second heat exchanger is used for patients over 80 pounds. Careful attention to consolidation of the equipment and shortening of the plastic flow lines should allow this to be reduced to 1,000 c.c., so that two pints of heparinized blood will suffice for priming the system.

There are a number of factors which contribute to providing an ideal operative field. (1) The isolated, drained heart is completely bloodless. When clamped off and drained, as illustrated in Fig. 2, a constant dry field is provided. Since no blood is flowing during the surgery, neither coronary flow, augmented bronchial return, nor the presence of a persistent left superior vena cava or a patent ductus arteriosus can obscure the field with blood. Only a minimal amount of blood need be aspirated when the heart is first opened, and this may be discarded or reused, as desired. (2) The operative field is completely quiet from cardiac arrest or fine ventricular fibrillation without the need for cardioplegic drugs, anoxia, or injections of cold blood. (3) No one but the surgeon who has performed open-heart surgery with a pump-oxygenator operating throughout the procedure can truly appreciate the blessing of having no equipment operating while he is working inside of the heart. He can devote his undivided attention to the surgery without any misgivings about the disastrous effects of equipment or personnel failures during the perfusion. Also, during the cooling and rewarming period, he is not busy doing surgery and can devote his attentions to this part of the procedure if necessary. (4) Since the heart is not beating and the aorta is clamped off throughout the intracardiac procedure, there is minimal danger of air embolism. We have seen no evidence of air embolism in these 27 cases. Careful filling of the chambers, as well as the vents provided by the right and left atrial catheters, further reduces this danger at the completion of the surgical repair.

An additional feature worthy of mention is that more time can be provided for further surgery, if required, at the completion of the safe period for circulatory arrest. Re-perfusing and recooling until the venous blood again becomes arterialized and the temperature reaches the desired level makes it possible to extend the period of circulatory arrest by a significant amount. In 2 cases of severe infundibular obstruction, when rewarming was started after completing the infundibulectomy and closing the right ventricle, bright red blood streaming out of the right atrial catheter indicated the presence of an undiagnosed atrial septal defect. These patients were recooled and perfused briefly, following which their atrial septal defects were closed and they made uncomplicated recoveries. One other patient with total anomalous pulmonary venous drainage and a large atrial septal defect was re-perfused and recooled between correction of the venous drainage and closure of the atrial septal defect.

COMMENT

Many modalities and techniques are available for the successful intracardiac repair of congenital cardiac defects. Deep hypothermia has been used by us successfully on a variety of such lesions and appears to offer definite advantages. For some lesions we will still probably desire a beating heart and the use of a pump-oxygenator. Further experiences should delineate more clearly the role of deep hypothermia and the patients who will profit most by its use.

REFERENCES

1. Gordon, A. S., Jones, J. C., Luddington, L. G., and Meyer, W. B.: Deep Hypothermia for Intracardiac Surgery: Experimental and Clinical Use Without an Oxygenator, Am. J. Surg. **100**: 332, 1960.
2. Drew, C. E., and Anderson, I. M.: Profound Hypothermia in Cardiac Surgery: Report of Three Cases, Lancet **1**: 748-750, 1959.
3. Drew, C. E., Keen, G., and Benazon, D. B.: Profound Hypothermia, Lancet **1**: 745-747, 1959.
4. Kenyon, J. R., Ludbrook, J., Downs, A. R., Tait, I. B., Brooks, D. K., and Pryczkowski, J.: Experimental Deep Hypothermia, Lancet **2**: 41-45, 1959.
5. Shields, T. W., and Lewis, F. J.: Rapid Cooling and Surgery at Temperatures Below 20° C., Surgery **46**: 164-174, 1959.
6. Brown, I. W., Jr., Smith, W. W., and Emmons, W. O.: An Efficient Blood Heat Exchanger for Use With Extracorporeal Circulation, Surgery **44**: 372-377, 1958.
7. Bigelow, W. G., Lindsay, W. K., Harrison, R. C., Gordon, R. A., and Greenwood, W. F.: Oxygen Transport and Utilization in Dogs at Low Body Temperature, Am. J. Physiol. **160**: 125, 1950.
8. Gollan, F.: Electrolyte Transfer During Hypothermia *in* The Physiology of Induced Hypothermia, edited by R. D. Dripps, Washington, D. C., 1956, National Academy of Science—National Research Council, pp. 37-41.
9. Pierce, E. C., Dabbs, C. H., Rogers, W. K., Rawson, F. L., and Tomkins, R.: Reduced Metabolism by Means of Hypothermia and the Low Pump Oxygenator, Surg. Gynec. & Obst. **108**: 339-352, 1958.
10. Young, W. G., Jr., Sealy, W. C., Brown, I. W., Jr., Smith, W. W., Callaway, H. R., Jr., and Harris, J. E.: Metabolic and Physiologic Observations on Patients Undergoing Extracorporeal Circulation in Conjunction With Hypothermia, Surgery **46**: 175-184, 1959.
11. Lesage, A. M., Sealy, W. C., Brown, I. W., and Young, W. G.: Hypothermia and Extracorporeal Circulation, A. M. A. Arch. Surg. **79**: 607-613, 1959.
12. Sealy, W. C., Brown, I. W., and Young, W. G.: A Report on the Use of Both Extracorporeal Circulation and Hypothermia for Open Heart Surgery, Ann. Surg. **147**: 603, 1958.

DISCUSSION

(PAPERS BY SCANNELL [PAGE 723], MULDER [PAGE 731], HARKEN [PAGE 744], KIRKLIN [PAGE 763], MORROW [PAGE 776], GORDON [PAGE 787], AND THEIR ASSOCIATES)

DR. DWIGHT F. HARKEN, Boston, Mass.—About 10 years ago Dr. Laurence B. Ellis realized that we were going to perform a lot of operations for mitral stenosis, so he set up a program for following these patients. He and his associates with the help of one to three full-time workers have carried this study through the years. I'd like to say a few things about this follow-up. This work has been reported periodically and largely supports Dr. Scannell's comments.

We are now in our third thousand operation on the mitral valve. The follow-up discussion today involves the first thousand.

The mortality rate has not been dissimilar to Dr. Scannell's. In ideally selected patients, the Group III patients of our classification, there was a mortality of less than 1 per cent. In the Group IV category, it was 21 per cent. Dr. Scannell used the medically treated patients of Drs. White, Bland, and Sprague for comparison with his surgical results.

We used Olesen's medical series. The series of Drs. White, Bland, and Sprague is quite similar to Olesen's and to current medical treatment. At the end of 9 years, 75 per cent of the Group III patients (those with predominantly progressive pulmonary symptoms) were alive, whereas there was only 40 per cent survival in a similar group treated medically.

Of those in refractory congestive failure (our Group IV), 60 per cent of the surgically treated patients survived after 9 years, whereas all of the medical control group were dead within 8 years. And gentlemen, survival is only a cold parameter of living, for among the surgical survivors there was much more happiness, comfort, and rehabilitation. The implications in patient selection are obvious. They should be operated upon when they are in Group III which has less than 1 per cent mortality and high rehabilitation rates.

Now I should like to take a slightly different point of view to illustrate a point that I'm sure Dr. Scannell has made, but I should like to make it in a slightly different way.

We don't have to prove to ourselves that surgery is useful, but we do want to know if and why these people deteriorate after being improved by surgery. In our first thousand patients, Dr. Ellis' group finds that there are 228 patients who are now, or were at the last review, less well than at some other time since operation.

Now this doesn't mean that surgery wasn't worth while, or even lifesaving, but 228 of them are now less well than they were at some time since operation. This is the important group for us to scrutinize to appreciate a point of view that Gordon is trying to emphasize.

There were four dominant overlapping factors responsible for this deterioration, and I emphasize "overlapping." The first and overwhelming one was what we would now term unsatisfactory initial operation—more than half the patients; second, recurrent rheumatic fever; third, mitral insufficiency prior to or caused by operation; fourth, multivalvular disease.

As for the unsatisfactory operation, this thousand was operated before we recognized the importance of opening the posteromedial commissure. Dr. Bailey has suggested the right thoracotomy to make this possible. I move around to the opposite side of the table in the standard left thoracotomy. Logan, Tubbs, Gerbode and others use a transventricular valvulotome. It is not so important how it's done as it is to recognize what must be done, and do it. Thus, I think we now take care of this factor in deterioration by more satisfactory initial (or secondary) operation.

The second factor, rheumatic fever, probably is less significant since the advent of routine permanent prophylaxis with antibiotics and the appropriate use of steroids. When this group was operated upon, routine permanent penicillin prophylaxis was not practiced.

The other two categories, mitral insufficiency and associated valvular disease, resolve themselves into better diagnosis and the appropriate rather than the inappropriate operations. When this group of patients was operated upon we had less satisfactory clinical and laboratory diagnostic techniques (left heart catheterization, etc.) and no adequate techniques for dealing with some of the lesions. There was no good operation for aortic stenosis and mitral insufficiency, much less aortic insufficiency, so we simply did the best we could with the mitral stenosis and hoped that the associated disease was insignificant. That can be changed now.

In conclusion, most causes of deterioration can be handled better now. Most importantly, we cannot only operate better initially but, by using the same principles at reoperation with an operating tunnel, we can make reoperation just as satisfactory as the modern improved primary procedure with bicommissural and subleaflet valvuloplasty.

In the light of this knowledge, when I hear talk about routine open operation for initial or reoperation, it worries me. Just because we have a pump-oxygenator, we don't have to use it. We don't have to use a bulldozer to mow the lawn. You reserve the bulldozer for the stumps, and if you use the mower well, it's surprising how few stumps there are.

DR. SCANNELL (Closing).—I don't know whether I should just close or ask Dr. Harken if I might discuss his paper.

Obviously, there is considerable agreement. I think the important thing for us to decide is: how to know that the stumps are going to be there, and use an open approach the first time around under these conditions, which I rather favor.

Second, it is important to recognize the break in compensation that occurs in these patients and, when they are getting into trouble, reoperate early.

I did not have time in the original paper to talk very much about this problem of reoperation. As we see it, we have been too reluctant to readvise surgery. We have operated upon patients after their lungs were badly damaged with severe arterial disease and, under these circumstances, end up with an excellent valve—but at autopsy.

DR. CHARLES P. BAILEY, New York, N. Y.—In view of the skepticism that greeted my first presentation of a somewhat similar technique to this Association 2 years ago, I am delighted to see that Dr. Mulder and others have done this type of operation and are getting good results.

(Slide) In fact, as I remember, the procedure was referred to as an "explosion in a cement factory" on that occasion.

(Slide) Over the past several years we have been doing open-heart surgery for acquired aortic stenosis. We did use hypothermia alone in some patients but gradually have come to depend upon complete bypass with coronary artery cannulation and perfusion, with or without the addition of hypothermia. We have had a mortality as shown in this latter group in 52 cases of 11.5 per cent. Generally the postoperative results have fitted in well with the later ones shown by Dr. Mulder.

(Slide) Louis Gross in 1936 showed the double layer make-up of the thin, transparent aortic valve (laminum spongiosum and laminum fibrosum), which permits the application of such a technique as Dr. Mulder has so beautifully demonstrated. However, the lesion, as he pointed out, may not be limited to the concave aspect of the valve, although it usually is.

It is true that one should mobilize the cusps by separating the commissures whenever feasible. In this photograph, this cusp, however, can be seen to be a shallow one, and this one is even more shallow. Had we done an open operation in this case, it might have been well to mobilize only this deeper cusp, to have separated it at the commissures, allowing the remaining two to be joined in a rigid shelf which would support itself if only by virtue of its attachment to two thirds of the aortic circumference. A good clinical result will be obtained even if only one cusp functions well.

(Slide) This slide merely shows the reparative technique that we have used in preference to using a plastic or prosthetic cusp. A segment of free aortic wall is used to create a new cusp after excising an extremely damaged one.

I have a one-minute movie which can be shown, demonstrating our technique.

DR. DWIGHT C. MC GOON, Rochester, Minn.—Dr. Mulder has nicely demonstrated his enthusiasm for the surgical treatment of calcifying acquired aortic stenosis, and, of course, Dr. Bailey shares that enthusiasm with him. So also do I.

I think it is understandable that when a condition for which there was no suitable treatment until 2 or 3 years ago becomes amenable to the surgeon, we should be enthusiastic about the prospect. Perhaps future follow-up studies will show that our optimism is not warranted, but perhaps also by that time newer techniques will have become available.

You recall that Dr. Kirklin reported upon 14 of our patients—the first 14—some time ago, among whom there was one early death and one late death. The series has now increased to 41 patients, with the mortality rate remaining at approximately 10 per cent. The only feature of the original technique which might be different in view of contemporary developments is that we used normothermia throughout the series. We have merely cross-clamped the aorta, and usually have begun to work on the left coronary cusp first. After

15 or 20 minutes have passed, work on this cusp is completed, and we then begin perfusion of the left coronary artery with about 400 c.c. of normothermic blood per minute. The heart very rapidly resumes its action, and usually it is possible to continue that perfusion throughout the remainder of the period of repair of the valves.

I think the most important feature of the technique is to be able to work in an unhurried manner, and not to feel the press of time. Work on the valves not uncommonly may continue for an hour or more.

By this means, when the aortic clamp is removed, the heart is oxygenated and as it has been vigorously contracting for a prolonged period, it is quite able to take over the circulation, even if a minor degree of aortic insufficiency should be present.

DR. JOHN F. BURKE, Boston, Mass.—We too have been interested in acquired aortic stenosis at the Massachusetts General Hospital, and have now done 42 operations; the last 25 patients have survived.

(Slide) We have noted that the more of these operations we do, the longer it takes us. We have used moderate hypothermia without coronary perfusion and, although we do not mean to imply that cardiac ischemia is good cardiac muscle, it is obvious that there are other factors which are perhaps even more important than perfusion of the coronaries. We believe one factor is an excellent restoration of valve function. Although coronary occlusion and ischemia was carried out in our cases, the results became better as we took a longer time to achieve a valve that would function almost perfectly.

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—I would like to draw your attention to one point.

Dr. Scannell said that if there was a gradient over the valve of less than 50 mm. Hg, he did not think that these patients were candidates for surgery. I think that in the case of aortic stenosis one should not only evaluate the gradient when the patient is resting. One should also evaluate the gradient at work and after work.

We have had patients with gradients as low as 20 or 30 mm. Hg after measured work. At rest, with one catheter in the left ventricle and one in the ascending aorta, the gradient might well rise to well above 100—up to 200 mm. Hg, and I think these patients are candidates for surgery as well.

DR. MULDER (Closing).—I should like to emphasize one point that we think is very important; namely, that it is better to start the dissection of the calcium at the base of the cusp and move centrally, rather than cutting blindly across the top of the encrustations.

With regard to Dr. Crafoord's remarks, we would certainly agree that the cardiac output varies under certain circumstances, and that this may alter the gradient measured across the aortic valve. We have operated on 1 patient with a low cardiac output and a gradient of less than 50 mm. Hg. He has had a very good result which suggests that in those patients with a low output, the demonstration of a gradient, even though less than the arbitrary 50 mm. Hg, may still be indication for operation.

DR. DONALD B. EFFLER, Cleveland, Ohio.—At the 1958 meeting of this Society in Boston, we discussed the need and our interest in prosthetic replacements of the aortic and mitral valve. Since that time Dr. Willem J. Kolff of the Cleveland Clinic Foundation has devoted considerable energy toward prosthetic valve replacement. Utilizing an excellent laboratory and applying the patience and energy characteristic of the indomitable Dutch, he has explored many facets of this complex field. I would like to show a short movie that demonstrates a number of significant experimental observations.

This is a Teflon mitral valve replacement; the dog died 10 days after operation. Viewed from the ventricular side it is self-evident that the prosthesis has become a thrombus factory. The animal died of multiple emboli.

This specimen is another mitral prosthetic replacement. The animal survived 4 weeks after operation and then developed a classical picture of mitral stenosis. This is not an isolated experience; unless the animal receives anticoagulants or fibrolysin materials, stenosis inevitably occurs in this type of valve.

This is an aortic monocusp replacement—the animal died with a large saddle embolus. The monocusp (polyurethane in this instance) contains an impaction of thrombus 14 days after insertion.

Dr. Kolff has worked with most of the available plastic materials and used all conceivable variations including the woven, knitted, smooth, and roughened types. The record of initial animal survivors after complete valve replacement has been excellent—the long-term survival rate is discouraging.

The concluding specimen is of particular interest. A Teflon strip has been fixed to the right atrial septum and dropped through the tricuspid valve where it is attached to the apex of the right ventricle. All dogs survived this simple procedure, but all succumbed at a later date when the atrial chamber follows the prosthetic strip and invaginates into the tricuspid valve in a manner reminiscent of a toilet plunger. Apparently there is an unexplained taxis phenomenon here.

The purpose of this film is obvious. It is not our desire to be distributors of sodden blankets. We all know that there are many perplexing and discouraging problems in this particular field—that, for the present, clinical application of prosthetic valve replacement at either the mitral or aortic level must be undertaken with considerable caution. For the present it is our policy in the surgical treatment of aortic and mitral valve disease to do the best we can with what the patient has. This policy will remain in force until we can provide our patients with better prosthetic valves than are available to us now.

DR. EARLE B. KAY, Cleveland, Ohio.—Whereas not over 10 to 15 per cent of the patients with mitral regurgitation require valvular substitution, it is our opinion that the majority of patients with aortic regurgitation and at least 50 per cent of patients with highly destroyed calcific stenotic valves will require valvular replacement for optimal correction.

The success of aortic valvular surgery is dependent upon three factors: (1) complete correction of the insufficient valve which assures, rather than hopes for, maximal or optimal valvular function; (2) proper myocardial support and management during and immediately following perfusion, and (3) a left ventricular myocardium that will tolerate the operative procedure.

During the past 4 years we have, as many of you also have, gone through the many intervening stages of partial or palliative correction of the insufficiency employing valvular addition, monocusps, plication, or excision of the noncoronary cusps, and the many other variations in techniques and have been impressed with the fact that, although occasional spectacular results were obtained with the above techniques, we were never assured at the time of the operative procedure that adequate or ideal correction had been accomplished.

Ideally, one must be assured at the time of the operative procedure that the insufficiency is entirely corrected and that no stenosis is produced. In our opinion this can only be accomplished by valvular substitution with a plastic tricuspid valve.

One hears the criticism that perhaps there is insufficient knowledge of the destiny of such valves but in actuality the only difference between a complete tricuspid valve and a monocusp or plastic used in valvular addition is the degree and design rather than the substance. The majority of the plastics appear well tolerated and have been used in various forms for 2 to 3 years. Furthermore, a tricuspid valve assures deep sinuses of Valsalva and proper coaptation of the leaflets. The high operative mortality in the past has been due to incomplete valvular correction and insufficient myocardial support and management during the procedure.

In regard to myocardial support, we have all had what we felt was a satisfactory correction of the insufficiency only to be confronted with a heart difficult to start, or to fail later because of the prolonged anoxia or inadequate myocardial oxygenation occurring during the period the aorta is cross-clamped. We have employed all of the current techniques for myocardial management during the period of cross-clamping the aorta and continue to feel that direct coronary artery perfusion, as described to this Society 2 years ago, is superior to other techniques if operating periods up to 1 or 2 hours are required. Shorter periods of aortic cross-clamping are well tolerated with cardiac arrest by perfusion of ice-cold Ringer's solution. Recently we used the combination of cardiac arrest by coronary perfusion of oxygenated ice-cold Ringer's solution to cover the 10- to 15-minute period to establish coronary cannulation, followed by direct coronary perfusion with oxygenated blood during the insertion of a plastic tricuspid aortic valve. The postoperative recovery of this patient was the most benign of any operated upon. We continue to use both left and right atrial decompression during the operative period as well as during the immediate post-perfusion period.

DR. WILLIAM H. MULLER, JR., Charlottesville, Va.—I, too, agree that in repairing the valves in patients with hypertrophied, dilated ventricles one must relieve the insufficiency without increasing the resistance at the site of the valve.

We make every effort to use the patient's own tissues in repairing aortic insufficiency but, in some instances, this is not possible and we utilize leaflet replacement or leaflet extension, and also total valve replacement.

I would like to present a very short filmstrip demonstrating a Teflon valve that we inserted in an 18-year-old patient who had aortic insufficiency as a result of bacterial endocarditis.

The extreme pulsation of the aorta is apparent. The aorta was clamped and an incision was made into it in a way similar to that demonstrated by Dr. Mulder. The posterior leaflet was completely absent except for a small tag, and there were multiple perforations in the two remaining leaflets. It was decided to replace the valve totally in this patient.

We, therefore, excised the remaining leaflets, leaving a small portion along the rim to which the prosthesis could be anchored. The sutures were placed through the tough, thick annulus, and also in the prosthetic valve. (The prosthetic valve had a Teflon cage covered with Teflon netting. The inside is polished, and the leaflets are knitted Teflon with polyurethane-treated edges. The cage is cut out to accommodate the region of the coronary artery orifices.)

The valve was positioned and additional sutures were placed around the top of the valve. We utilized coronary perfusion of the left coronary artery, and we have been using it in the right coronary artery while we are closing the aorta. The pulsations of the aorta which were formerly present were greatly diminished.

This patient was operated upon about 5 weeks ago and is doing satisfactorily. We don't believe that this valve is the answer, but we hope that it's a step in the right direction. Many questions must be answered regarding the long-term fatigue properties of Teflon. Certainly, stiffening of the leaflets as Dr. Effler showed a moment ago and a number of other points will have to be considered. None of the patients in whom this valve was used has presented any evidence of emboli. We have totally replaced the valve in 5 patients, 2 of whom are still doing satisfactorily; 1 early and 1 about 18 months since operation. All of these patients had severe aortic insufficiency and, at the time of operation, it was believed that total replacement was necessary to enable them to survive the perfusion.

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—I want only to bring out a few points that haven't been brought out relative to the use of autogenous tissue in aortic insufficiency.

Aortic insufficiency may at times be caused by a prolapsed cusp. (Slide) The anatomy of prolapse is depicted diagrammatically here. This cusp prolapses because of weakness of the supporting strut made up by the spongiosa, annulus fibrosa, and aortalis as they form the strut for the coapting ridge, which in its center contains the nodulus arantii, all three of which must meet at a single point to produce a competent valve.

(Slide) This weakness of the strut support to the cusp close to the aortic wall produces the sagging of the lunula and a disalignment of the noduli arantii on closure of the valve in diastole, so that it becomes incompetent.

(Slide) This slide diagrammatically depicts the physiology of the prolapsing valve on diastole. This incompetent space remains. Therefore, the point of attack is this anatomic area of the valve close to the aortic wall, in order to splint the sagging lunula on the well lunula by a supporting strut made with a free arterial graft taken from the internal mammary artery.

(Slide) The technique shown on this slide has been utilized in a 46-year-old man with severe aortic insufficiency and a prolapsed cusp, who was operated on 18 months ago and who, at the present time has improved radial arterial tracings. He had not worked for a year and a half, and he has been able to work for the past year. Diastolic pressure was raised from 20 to 60, and his cardiac size is smaller than it was preoperatively. The strutting material was autogenous arterial segment taken from the internal mammary artery.

We have found in dogs that free arterial segments, when planted in the myocardium, the endocardium, or in the valve itself, are well tolerated, and in periods of time up to 16 months these free arterial grafts tend to harden and become supportive.

DR. HARKEN (Closing).—Dr. Effler's observations are timely, and we too have feared stiffening and contraction of materials that become organized as scar tissue. This is why we have been reluctant to use wettable materials. However, Dr. Muller's experience suggests that some of our fears are unnecessary. I hope he's right. It would simplify the problem. At least our caged-ball valve does not have some of these potential disadvantages. Perhaps it will have disadvantages of its own.

I agree with most of the fine points made by Dr. Kay and appreciate his emphasis on the importance of avoiding stenosis. Here again you get the undertone of alarm in all of these discussions about aortic insufficiency with its dilated ventricle in failure.

Dr. Muller's brilliant cases warrant his enthusiasm and our commendation. I noticed that he was perfusing only one coronary artery. One cannot argue with success, but I believe that, if one is good, two are better. A substantial part of the left ventricle is perfused by the right coronary artery, therefore we should perfuse it as well as the left.

Dr. Goldman's comments about the prolapsed cusp represent excellent observations. It was no coincidence that his diagram showed prolapse of the noncoronary bearing cusp. As a mater of fact, that concept was the basis for our circumclusion operation, although I admit that it has been less than dramatically successful. It was also the basis for the first anatomic drawing that I showed today in which the base of the aorta was dilated and the neutral cusp had prolapsed downward and backward. Aortic insufficiency in these situations is a two-dimensional affair, one transverse and another longitudinal. I would be a little wary of producing stenosis whenever suturing commissures together, but obviously he was wary and successful.

As usual, a paper presented to this Society has flushed out a lot of valuable information. From you I have learned a lot. As the expression goes, thanks for letting me bring the can opener to the picnic.

DR. HENRY T. BAHNSON, Baltimore, Md.—This is the usual loaded, concise, forthright Kirklin presentation on which I would like to compliment him and make a few comments about one small segment of patients with ventricular septal defect; namely, those with aortic insufficiency.

In many if not most patients with ventricular septal defects there may be some degree of aortic insufficiency, which will vary from a smidgen to a sizable stream. Usually

this can be ignored. It is not clinically detectable, and simply bothers the surgeon when he is attempting to repair the defect.

When there is significant clinical aortic insufficiency, however, we believe that the basic mechanism is that shown in this first slide (slide), namely, a prolapse of the right coronary cusp, shown here, and possibly a segment of the adjacent noncoronary cusp.

We have not been successful in attempting to coapt the commissures in these patients, and believe that, although the original trouble probably arises from the prolapse of the base of the leaflet, that the trouble we need to treat is in the greater length of the free edge, which actually prolapses beneath the adjacent cusp.

(Slide) We have treated this, as shown here, by simply reefing the free edge, in 3 patients, with excellent results in 2. I think the movie will show a specimen from the third patient. We obtained this specimen because I was, perhaps, obsessed with the idea that one could treat this by narrowing the aortic annulus, closing the defect in an improper fashion. This partially reopened, and the patient died of insufficiency.

I think this specimen demonstrates that the important phenomenon is reefing the free edge. This is viewed through a Lucite chamber in which water is put upon the valve, so that the pressure is constantly equal to the systolic pressure, and the valve is kept sealed. You notice that the cusps are at least together.

Here is the reefing. Here are sutures used below to close the ventricular septal defect. The sutures are removed, and you see that it makes no difference, really, in the competence of the aortic closure. This patient had a normal blood pressure after closure, and for a day and a half; then the pressure increased—I believe because of the tear in the base of the leaflet through which you saw the clamp come then.

Now, you will notice that the cusp had prolapsed down beneath the adjacent cusp. Notice how thin the base of this leaflet is where it has prolapsed through the ventricular defect. It does very little good to close the defect and buttress up the base of the valve. Again, the trouble is in the free edge.

(Slide) Here is the specimen after it has been fixed, so that the nearly normal arrangement can be seen. Here is the right coronary cusp prolapsing through the defect. Actually, at operation this almost sealed off the defect, so that there was very little leakage of blood through it. Notice how this has actually become detached from the edge of the ventricular defect.

Looking at this from above again, notice how the valve prolapses down, because of lengthening of the free edge. The difficult thing is to determine how much reefing one should do, and what we have done is to measure the diameter of the aorta with the aorta distended before we open the heart, and then to measure the length of the free edge of the cusp, and it is easy to see, on working with the valves, that the length of any free edge should be approximately the same as the diameter of the aorta, if the confluence of the cusps in the center of the aorta is to be obtained.

DR. EARLE B. KAY, Cleveland, Ohio.—We have been very pleased with the results obtained in the surgical correction of ventricular septal defects in a series of nearly 100 patients operated upon during the past 4½ years. Until recently we have had no deaths in any patient with pulmonary hypertension below 75 mm. Hg. About 2 months ago we had our first death in this group as a result of a staphylococcus-resistant infection.

During the past 3 years no patients have died, except for this one just discussed, regardless of the degree of pulmonary hypertension as long as there was still a left-to-right shunt. We have not operated on any patients with Eisenmenger's syndrome.

During the 4½ years that comprise this experience, there has been no instance of complete heart block. There were three temporary heart blocks that were immediately converted with Isuprel during the period of re-establishing the heartbeat at the conclusion of the surgical correction. We have not operated upon patients under 1 year of age, and, as far as I know, no patients have died during the last several years with conservative

treatment during this early period of infancy. In any event, it is our opinion that the risk of medical management during the first year of life carries a lower mortality than when attempts at surgical correction are made.

Initially, we did not employ cardiac arrest, but during the past 3½ years probably over 90 per cent of the patients have had cardiac arrest with potassium chloride. Over 90 per cent of the patients have had closure of ventricular septal defect using a compressed Ivalon patch with interrupted silk sutures. We have been very careful in taking multiple small bites around the ventricular septum and probably this is the reason that we have not encountered any evidence of heart block.

We have had five recurrences during this time, all occurring during the first year. The first two were as a result of using non-compressed Ivalon, in which the pores increased in size. The other two were in patients who had closure without an Ivalon prosthesis. The fifth was in a patient with a Swiss cheese type of defect.

All 5 patients have now been reoperated upon. Four of the defects are completely closed, and the 1 patient with the Swiss cheese type of defect still has some partial opening.

DR. KIRKLIN (Closing).—Certainly, Dr. Bahnson has demonstrated better than anyone previously the anatomy of aortic incompetence in patients with ventricular septal defect. We can confirm his belief that it does no good merely to repair the ventricular septal defect. In the first case that we had some time back, we merely repaired the ventricular defect, thinking that that would push the aortic leaflet up where it should be and relieve the incompetence. The patient has survived, but still has a significant aortic valve incompetence.

I can only envy Dr. Kay and his results, and perhaps differ with him in one small point. I doubt that we can confidently assume that conservative management for the sick small infant in heart failure from ventricular septal defect will allow us to operate on him at the age of 2 years. In 1957 we banded some of these infants and treated some of them medically. Dr. DuShane in our clinic treated them carefully and diligently. Twenty-eight per cent of that small group died before they reached the age of 1 year. They did not die in failure in the hospital. Usually they went home, suddenly acquired a respiratory infection, and had overwhelming bronchopneumonia before they could be returned to the hospital.

DR. BRUCE JOHNSTON, San Francisco, Calif.—The few remarks which I have to make are merely to reinforce, I hope, the thesis which Dr. Morrow has so well developed.

There are several reasons why we have adopted extracorporeal circulation as the technique to allow us to repair secundum defects, but they are all linked essentially with the fact that we do not feel that the 6 or 8 minutes which hypothermia alone will give us is adequate in our hands for a satisfactory repair.

We have an early experience with some 40 cases which we closed by the external suture technique, and in 5 of these patients there was a very large residual shunt. They were all adults. The defect at the time of the first operation had always been large, and this indicated quite clearly to us that a certain number of these secundum defects would require some prosthesis in order to assure complete correction.

Furthermore, we find that in our series some 12 per cent of patients have anomalous venous return. Admittedly, a small number of these patients can have a correction performed without inserting a prosthesis, but the majority certainly do require a patch.

In all, we find that 10 per cent of our patients needed a patch of some sort. And so, for the last 104 patients we have used extracorporeal circulation, and with the most recent 45 patients we have used the lung recently described by Osborne, Bramson, and Gerbode.

We have had one death in this group of 104 patients, a child who was in severe cardiac failure at the time of operation, and we have good reason to believe that all the others are satisfactorily corrected.

DR. GEORGE H. A. CLOWES, JR., Cleveland, Ohio.—Dr. Morrow has just emphasized the need for doing very accurate surgery, for one reason—that is, to effect a complete repair. Dr. Scannell and other speakers this morning have talked about the quality of the repair of a valve and the dangers of regurgitation. I would simply like to emphasize why it is that certain people produce beautiful results by rendering the heart capable of responding in normal fashion to the trauma of operation.

(Slide) The cardiac output during the immediate postoperative period seems to follow a pattern in patients after thoracotomy. You will notice that there is a tremendous rise of cardiac output at the time the endotracheal tube is removed and, if the cardiac output goes up then, one can be sure that the patient will do well postoperatively. During the first week the cardiac index remains about 30 per cent above the basal value. Note that the venous pressure is up only during the period of the operation and the anesthesia.

(Slide) Now, for comparison here is a series of 13 patients who had cardiac operations, studied in the same fashion, who survived. Notice that they started with a lower cardiac index, but it came up at the time of extubation. Their cardiac output actually went up after the cardiotomy had been completed. Then there was a short period of depression during the first few hours. Thereafter it remained up as in the rest of the thoracotomies.

(Slide) This will show you the behavior of 6 patients who died. Four of these were patients who had cardiac operations, one of them being an aortic valve procedure. The cardiac output started with a low level, went down during the period of operation, but failed to go up at the end of the operation. Not one of these patients reached the preoperative index.

Dr. Boyd and his associates from Johns Hopkins showed the same thing last year, and other people have emphasized this, but I believe this will serve to emphasize why we must do a complete repair on these various cardiac abnormalities. If the myocardium is damaged by failure to perfuse the coronaries, or for other reasons, then we see a similar situation. Patients who fail to respond in this normal fashion die in a state of metabolic acidosis.

DR. HENRY SWAN, II, Denver, Colo.—We have continued to use the open closure of atrial septal defects with hypothermia alone, and we have had experience now with about 165 patients in whom this operation has been done.

We also agree with Dr. Johnston that about 12 or 13 per cent of these patients have some aberrant drainage of some type, usually associated with the very high, or sinus venosa, defect. We have had 22 such patients.

We have used hypothermia at 30° C. rectal temperature. We limit our occlusion period strictly to 6 minutes, and will not exceed that amount of time, but we do not hesitate to do two or three occlusions, and thus one can get 15 or 18 minutes of operating time, if so desired.

In our last 125 patients we have had one death.

The results have been documented by postoperative catheterization in 44 patients, largely in the earlier part of the series, as Dr. Blount is no longer doing postoperative catheterization routinely in this type of patient. Five of those 44 patients had evidence of failure to close completely the defect. Two of these were in the early group of patients when we were using interrupted sutures, and I think I spaced the sutures too far apart. Both patients were reoperated upon, and the residual hole was closed.

One was known at operation to have been left with a vein which was untransposed, so it was no surprise to find some residual shunt in that patient.

Two were actual failures of the technique. One of these was an adult patient with a very large heart and a very large hole, measuring 9 cm., and I feel that in this patient there was a complete breakdown of the suture line. An attempt was made to close it using cardiopulmonary bypass at another institution later on, but the patient failed to survive the procedure.

I feel that we could quite agree with you that in the adult patient with the very large hole we may well need to put in prosthetic devices, but so far at least in our experience we

have not used any prosthetic devices, and we think that it is quite possible to get accurate, successful closure at a very low risk using this technique.

DR. MORROW (Closing).—The object of one of these intracardiac exercises is to close the hole completely. We are not satisfied with any residual shunt after an open operation, and I don't think that we can detect small shunts by the conventional methods.

For example, among those patients undergoing postoperative catheterization a year or two ago, the oxygen method was being used in our laboratory at that time as well as the N_2O test. Four of 8 such patients had no detectable shunt using the conventional methods, but the residual shunt was demonstrated by the inert gas test.

With the open operation with bypass I think that Dr. Gilbert and I would probably use a prosthesis now in 4 out of 5 patients with atrial septal defect, and our average perfusion time in an adult with an atrial septal defect approximates 45 minutes.

DR. FRANK GERBODE, San Francisco, Calif.—Our early experience with potassium arrest taught us one thing, that it was advantageous to operate upon the quiet heart. However, in the past 18 months we have not used potassium arrest, and have turned to using general body hypothermia in combination with extracorporeal circulation, and during the past year we have used this technique in over 200 open-heart operations.

General body cooling has been accomplished with the use of a new, very efficient heat exchanging lung (slide), which was developed in our laboratories with the help of M. L. Bramson and Dr. John Osborn. Our lung has a relatively low priming volume, and has the entire casing of the inside of the lung free and exposed to a water jacket. This is a very efficient heat exchanging unit, and it doesn't require any separate cleaning, because it is isolated from the blood circulation.

We have employed various degrees of hypothermia in combination with extracorporeal circulation, and, as you can see, the vast majority of patients have been only slightly cooled, and ventricular fibrillation has not been induced.

However, for the more serious types of lesion with various types of acquired heart disease and tetralogy of Fallot, arrest has been induced by general hypothermia low enough to induce ventricular fibrillation. This permits a lower flow than is necessary under normothermic conditions.

This slide shows the rapidity with which a large patient can be cooled using this device. Rewarming is slightly slower, of necessity.

(Slide) We believe, too, that left atrial decompression is an absolute necessity for any patient who has a heart stopped by cold fibrillation or cold arrest. We first used an entry through the left atrial appendage but found this rather difficult to manage, and have now, in the last year, employed a catheter placed posteriorly in the portion of the left atrium which presents on the right side. If one passes a catheter through this area, you can in some instances thread it through the mitral valve and simultaneously decompress the left ventricle as well. This is put in place in the early part of the perfusion, and it stays there until the heart stabilizes on coming off the pump.

I believe the combination of general hypothermia with induced ventricular fibrillation with cold has resulted in patients who are metabolically better off during the postoperative period. We have documented this in various ways, but it has also been clinically apparent to all of us. As an example of the effectiveness of the technique, there were two operative deaths in the last 16 operations for cyanotic tetralogy.

DR. BENSON B. ROE, San Francisco, Calif.—We have enjoyed the technical and clinical advantages of both general and regional hypothermia in conjunction with our perfusions, and I think with considerable advantage to the patient, as Dr. Gerbode indicated. Indeed, I think that we could say that our postoperative pattern of acidosis has been converted to one of alkalosis.

I rise principally to submit a simplified technique which has versatility and which permits the use of a single heat exchanger for both cooling and warming, and for both regional and general hypothermia.

The first slide demonstrates the method we now use routinely, which permits us to decide whether regional hypothermia is necessary during the procedure without necessitating additional equipment. This is our heat exchanger set up with the clamp placed here, so that from the reservoir source a single pump carries the blood through the heat exchanger for general body perfusion to control body temperature which is routinely lowered to 30° C. or lower.

If cardiac arrest is desirable, then this "Y" in the system is opened and the already cooled circulation is then partially shunted past the heat exchanger for continued body perfusion. A smaller portion of the same pump output passes through the other side of the "Y," through the heat exchanger (now connected to an ice water tank) and thence into the coronary perfusion. The clamp can be reshifted for body warming afterward.

(Slide) This is a "saddle" needle which rests like a saddle over the aorta, with a curved needle designed to lie inside the ascending aorta, without harming the valve or piercing the posterior wall.

(Slide) This slide shows it in place. It is tied with a piece of umbilical tape around the aorta and across the saddle, and lies unattended out of the operative field.

DR. DONALD B. EFFLER, Cleveland, Ohio.—Scientific papers can be like old love letters in that the context written in all sincerity may be embarrassing when read years later.

On previous occasions, there have been references to a paper presented by Dr. F. Mason Sones, Jr., a member of the Department of Cardiology at the Cleveland Clinic. In this paper Dr. Sones cited the occurrence of myocardial necrosis at autopsy in patients who had succumbed following our early efforts in open-heart surgery. At the time of that publication, we did not understand the problems relative to fatal metabolic acidosis or the hazards of low flow perfusion; nor did we realize the myocardial changes that would occur in patients who had gross overdistention of the ventricles and the attendant damage to the pulmonary venous bed. The myocardial damage described in that paper is no longer a frequent finding in patients who do not survive operation whether cardiac arrest was employed or not. For the record I believe that we should deal with present knowledge rather than a misconception published almost 4 years ago.

Perhaps I have been stubborn in my continued use of elective cardiac arrest utilizing potassium citrate. It is still a very useful adjunct in surgery that utilizes extracorporeal circulation, but must be used properly and in selected cases. As we have reported many times in the past, adults and children alike do tolerate potassium arrest for periods of time that may exceed 60 minutes.

I agree with the authors that regional hypothermia is an extremely valuable adjunct, particularly in surgery of the aortic valve; whereas our technical methods may differ slightly, the basic approach is quite similar. I would like to emphasize again, however, the necessity for proper and adequate left heart decompression while the isolated heart is perfused, and in particular during the rewarming period before the left ventricle has regained its proper tone.

Ventricular fibrillation is almost invariably seen in patients who have had cold arrest. In patients who have had severe rheumatic heart disease with marked myocardial damage and enlargement, this can be a real problem in resuscitation. In an earlier paper Dr. Harken mentioned 1 case in which electric shock was employed over 20 times in an effort to convert ventricular fibrillation to an effective rhythm. Our experience has been similar and for this reason we make no effort to defibrillate the heart until we have brought its temperature back to a comparatively normal level. If ventricular fibrillation is still persistent, we have employed one small trick that is worthy of mention and that is the use of quinidine sulfate. Quinidine sulfate may be given by the Melrose technique, and its

action can be very prompt. We have employed it in large doses on occasion by direct injection into the extracorporeal circulation. When used in this manner, however, it may be conducive to rather severe hypotension even after an effective ventricular rhythm has been re-established.

DR. Å SENNING, Stockholm, Sweden.—There have been some deaths reported from cerebral damage after this type of procedure, and therefore I wish to discuss the conditions of the brain temperature. The brain temperature is dependent on many factors, the most important being the perfusion technique used.

If we are using the femoral artery for the arterial cannulation and are using flows through the heat exchanger or through the pump that are significantly smaller than the cardiac output, then the upper part of the body, including the brain, is perfused by "warm" blood from the heart itself.

(Slide) This slide shows an experiment where we have had a rather low flow through the double pumps and the heart has been in good action. The temperature in the brain is shown here (indicating), and the one in the esophagus and rectum are shown here.

The temperature in the brain falls very slowly compared to the temperature in other organs up to this point here, where the heart stops. At this point there is a difference in temperature of 12° C. Then the brain temperature also falls rather rapidly, but still here when it's 10° or 11° C. in the esophagus and the rectum, there is a temperature of about 20° C. in the brain.

(Slide) The next slide shows more or less the same as Dr. Gordon showed us in his excellent presentation. In this experiment the temperatures in the esophagus and the rectum have fallen to about 12° C., but the temperature in the brain is still 22° C. During an induced circulatory arrest (this time it was 50 minutes), the temperature in the brain goes up from 22° to 25.6° C. I don't think that a patient in similar condition could stand a cardiac arrest of 40 minutes.

We have to be aware of the possible difference in temperature between brain and esophagus and the rise in temperature during circulatory arrest.

DR. ROLLINS HANLON, St. Louis, Mo.—My comments are applicable to the last two papers; namely, selective hypothermia in cardiac arrest as contrasted with generalized hypothermia.

One of the reasons for uncertainty about the inocuousness of cardiac arrest has been the lack of critical assessment of cardiac function after arrest and ischemia. Studies which have been done by Dr. Willman and Dr. Howard and others in our laboratory bear on this problem.

(Slide) These are left ventricular function curves, with the upper curve showing function in the control period before hypothermic arrest. After arrest of the heart for 30 minutes in the dog, you can see that the ventricular function curve shows considerable depression of left ventricular stroke work in gram-meters. The temperature of the heart has been lowered to the point of arrest by the perfusion with cold blood, but with cessation of perfusion the temperature of the heart has drifted upward, as was emphasized by Dr. Gordon. With this elevation, the heart goes into ventricular fibrillation, followed by arrest; that is essentially anoxic arrest. Hence mere arrest by cold blood with subsequent rise in temperature exposes the heart to the same damaging effects that one gets, theoretically at least, from anoxic arrest.

(Slide) In contrast, if one attempts to keep the temperature of the heart down by internal and external lavage with cold solutions, you can see that ventricular function is good after arrest, although not as good as in the control period. This is a matter of how efficiently one cools the heart locally.

(Slide) The final slide indicates the use of total body hypothermia, so that the temperature of the heart really remains at 15° C. Under these circumstances with 30 minutes of arrest, the ventricular function after rewarming shows a curve that closely approximates the control valve.

We would like to stress the fact that if one stops the heart by local hypothermia, unless that cardiac hypothermia is maintained, the deleterious effects of anoxic arrest will become manifest.

DR. JOHN Y. TEMPLETON, Philadelphia, Pa.—We have done some work in the laboratory, as have many of you, I'm sure, with double pumps and the heat exchanger. We have, in general, used, I think, a flow which is probably somewhat larger than Dr. Gordon's having maintained it by addition of blood as required, and with this method the decrease in over-all body temperature is considerably greater. For example, muscle temperatures are somewhat lower than he has stated here.

I should like to show one slide (slide) to point out something about the carbon dioxide in this type of preparation. When one is using the intact lungs, one is using a gas exchanger which is a great deal more efficient than those which we are accustomed to use in the heart-lung bypass. This is an adult female patient who had an atrial septal defect, and was cooled to only 20° C. with this method. You will observe the very significant fall in the partial pressure carbon dioxide in the arterial blood, and the rise in the pH; this in spite of the fact that during this cooling period the patient was ventilated using a non-rebreathing system with 3 per cent carbon dioxide in air. You will notice that the PCO_2 of the arterial blood is quite close to the PCO_2 of the inspired air.

DR. GORDON (Closing).—There are many more questions which could be raised regarding the clinical application of deep hypothermia. Our own investigations in this area are continuing in an effort to resolve some of the remaining problems.

Early in our deep hypothermia experience we were concerned with the possibility of central nervous system damage. However, studies on over 60 dogs subjected to prolonged periods of circulatory arrest under deep hypothermia failed to reveal central nervous system damage when the duration of circulatory arrest did not exceed the limits defined in this report. Our experiences have been the same with the patients in this series. The absence of postoperative electroencephalographic changes, the absence of neurological findings, and the absence of behavior changes in these patients indicates to us that the central nervous system has been protected adequately.

As regards brain temperature, our experiments in dogs reveal that the esophageal and brain temperature parallel each other closely when the thermistor probes are properly placed. During circulatory occlusion, the brain temperature drifts up less than the esophageal. This probably results from the esophagus being surrounded by a large mass of warmer muscle and subcutaneous tissues, whereas, the brain is isolated and retains its temperature better.

Anesthesia is not required when the patient's temperature drops below 28° C. We have used 5 per cent carbon dioxide and 95 per cent oxygen as the inhalant during cooling and rewarming. There are a number of reasons which can be advanced for this use of carbon dioxide. Our primary reason is that, in the hyperventilated patient, the addition of carbon dioxide tends to preserve the base bicarbonate. This is important because there is always some degree of metabolic acidosis following rewarming.

At the completion of rewarming the mixed venous pH is usually in the range of 7.28. When an adequate blood pressure is maintained the pH is usually back to normal within 8 hours. None of these patients has required intravenous bicarbonate or lactate postoperatively to combat acidosis.

I hope that by the time of this meeting next year, many of you will have used deep hypothermia for intracardiac surgery and that we will have an opportunity to compare our experiences at that time.

MYOCARDIAL DYSFUNCTION AFTER CARDIOPLEGIA

An Experimental Study

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TODAY cardioplegia achieved with potassium citrate (the Melrose technique)[1, 2] is condemned as vociferously as it was enthusiastically acclaimed 4 years ago. Other cardioplegic agents and techniques have since been introduced and in turn received with varying degrees of enthusiasm. We share the clinical impression with others that induced cardiac arrest has often produced serious impairment of myocardial function. However, hypotension and circulatory failure cannot be ascribed solely to cardioplegia. The ventriculotomy and the hemodynamic adjustment required of the heart are important determinants. Because of the many factors involved in the success of a complicated open-heart procedure, the survival of the animal or patient has proved to be an unreliable or inadequate measure of the safety and relative value of a cardioplegic agent. Our investigation was undertaken in order more accurately to evaluate and quantitate different methods of elective cardiac arrest.

Sarnoff and Berglund[3] have shown that ventricular performance can be expressed as a function curve in which ventricular stroke work is plotted against the corresponding mean atrial or ventricular filling pressure. In their experiments, which did not involve extracorporeal circulation, the data were obtained by progressively elevating the atrial pressure with increments of blood transfused into the experimental animal. The resulting cardiac output was measured by means of a flow meter. Stirling, Stanley, and Lillehei[4] studied the effects of ventriculotomy upon right ventricular function using Sarnoff curves. They collected the necessary data by controlled venous inflow. We have employed a similar technique in our own studies.

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METHODS

Twenty-six mongrel dogs, averaging 9.5 Kg., were medicated with 4 mg. per kilogram morphine sulfate and anesthetized with alpha-chloralose and urethane, after induction with Pentothal sodium. Heparin was the anticoagulant used, 5 mg. per kilogram. Extracorporeal circulation with total perfusion was established for 15 minutes with a rotary pump and homologous lung oxygenator (Fig. 1). The perfusion circuit was then modified for controlled venous inflow and, at increasing flow rates from 600 to 3,000 c.c. per minute,

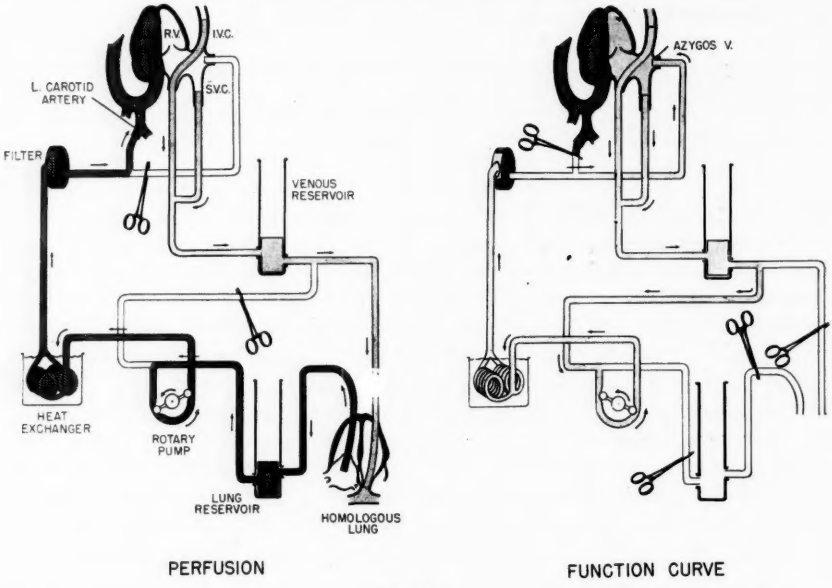

Fig. 1.

the mean right atrial, pulmonary artery, left atrial and aortic pressures were recorded. With pressures converted to centimeters of water, the following formulas yield ventricular stroke work:

$$\text{RV stroke work (Gm.-meters)} = \frac{\text{Flow (c.c./min.)} \times (\text{PA} - \text{RA pressure})}{\text{Pulse rate} \times 100}.$$

$$\text{LV stroke work (Gm.-meters)} = \frac{\text{Flow (c.c./min.)} \times (\text{Ao} - \text{LA pressure})}{\text{Pulse rate} \times 100}.$$

All function curves were determined at 35° C. The average maximum control stroke work for all left ventricles was 28.8 Gm.-meters, and for all right ventricles was 5.2 Gm.-meters. Function curves for each normal ventricle were constructed by plotting each filling pressure (mean atrial pressure) against the stroke work which it produced (Curves I for Figs. 2, 3, 4).

Total perfusion was then reinstituted and the heart arrested by one of the five techniques described later. After a 15-minute period of cardiac depression or arrest the heart was released and cardiac action resumed (with

defibrillation in 2 cases). Both atria were decompressed until cardiac action was adequate to prevent distention of the heart. Function (Curve II) was studied after 15 minutes of recovery perfusion, and again (Curve III) after an additional perfusion of 15 minutes (approximately 1 hour after arrest).

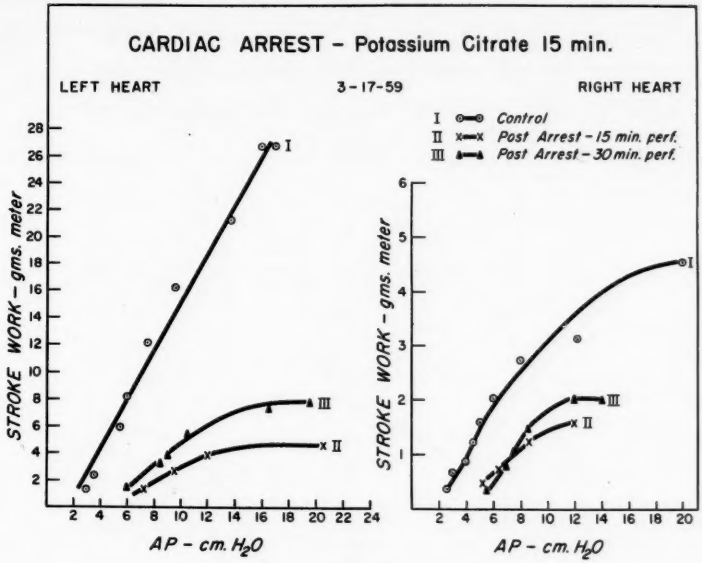
CARDIAC ARREST – Potassium Citrate 15 min.
LEFT HEART
3-17-59
RIGHT HEART
I Control
II Post Arrest – 15 min. perf.
III Post Arrest – 30 min. perf.
STROKE WORK – gms. meter
AP – cm. H2O
STROKE WORK – gms. meter
AP – cm. H2O

Fig. 2.

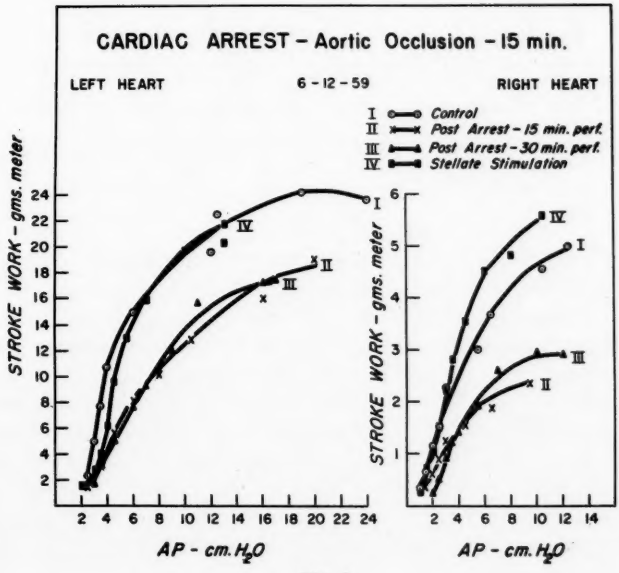
CARDIAC ARREST – Aortic Occlusion – 15 min.
LEFT HEART
6-12-59
RIGHT HEART
I Control
II Post Arrest – 15 min. perf.
III Post Arrest – 30 min. perf.
IV Stellate Stimulation
STROKE WORK – gms. meter
AP – cm. H2O
STROKE WORK – gms. meter
AP – cm. H2O

Fig. 3.

(In some experiments the effect of stimulating the isolated right stellate ganglion was evaluated [Curve IV].) The function curves thus obtained were then plotted together to form a family of curves produced by a single heart (Figs. 2, 3, 4).

Heretofore function curves of variously treated hearts have been compared by gross visual impression in regard to maximum peak, slope, and displace-

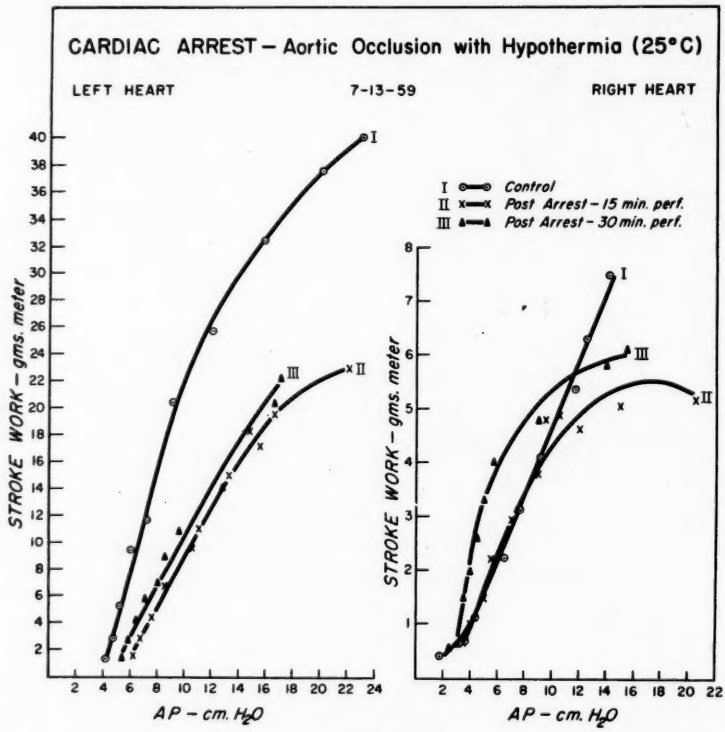
CARDIAC ARREST – Aortic Occlusion with Hypothermia (25°C)
LEFT HEART
7-13-59
RIGHT HEART
I Control
II Post Arrest – 15 min. perf.
III Post Arrest – 30 min. perf.
STROKE WORK – gms. meter
AP – cm. H2O
STROKE WORK – gms. meter
AP – cm. H2O

Fig. 4.

ment along the abscissa.[1-6] This comparison "by impression" does not allow statistical integration of several experiments. Every function curve represents its heart's functional capacity over the wide range of filling pressures by which it might be challenged. It is impossible by any single formula to assign to every function curve a numerical value which is quantitatively significant of function. However, the relative values of two curves in the same family may be expressed numerically as the average ratio between their stroke works over a range of atrial pressures. This average ratio is accurately expressed by relating the areas under curves delimited by properly selected ordinates (Fig. 5). In these experiments control curves are considered 100 per cent of ventricular function, and post-arrest curves are related to the control curves in terms of per cent.

RESULTS

Acetylcholine (10 mg./Kg.) achieved arrest promptly when injected into the proximal, clamped aorta of normothermic dogs. In 3 dogs the aorta was released after a 3-minute arrest, and in 4 dogs arrest was maintained for 15 minutes. Cardiac contraction returned promptly upon release of the aorta in all cases. Post-arrest function curves are expressed as per cent of control function as shown in Table I. The average values for per cent function shown in Table I are plotted in Fig. 6. Significant is the finding that marked depression of both ventricles occurs after acetylcholine arrest of either duration, but a more rapid recovery is found after the shorter arrest (Fig. 6).

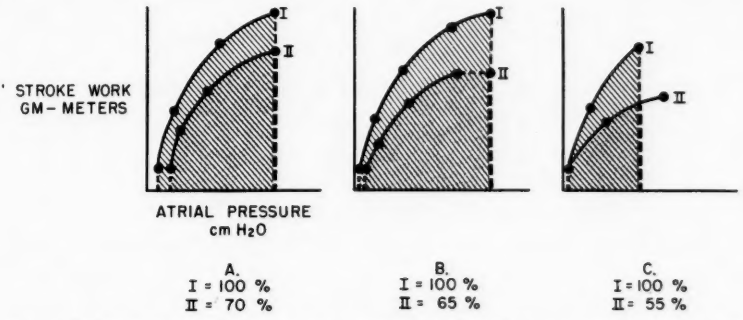

Fig. 5.—Hypothetical function curves demonstrating method of deriving curve area and per cent of function.

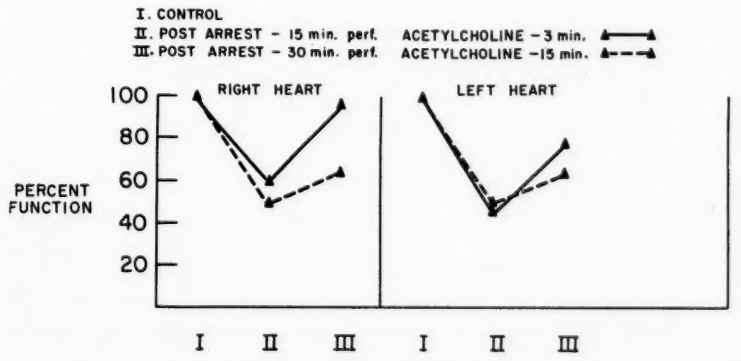

Fig. 6.—Delayed recovery of ventricular function by prolonged cardiac arrest.

Potassium citrate (2.5 per cent) produced immediate arrest in 7 normothermic dogs. In 3 animals, the aorta was released after a 3-minute arrest, while in 4 the arrest was maintained for 15 minutes. In all cases cardiac action resumed promptly. Function curves derived from these experiments were converted to areas and per cent of controls, and results are shown in Table II. Technical errors in the 3-minute arrest experiments produced inexplicable results, while 15-minute arrest experiments resulted in marked depression of both ventricles with partial, but very incomplete, recovery

TABLE I. ACETYLCHOLINE CARDIOPLEGIA

EXPERIMENT NUMBER	MAXIMUM STROKE WORK (GM.-METERS)						PER CENT FUNCTION					
	RIGHT			LEFT			RIGHT			LEFT		
	I	II	III	I	II	III	I	II	III	I	II	III
AC-3 min-1	3.5	3.9	4.4	30.0	25.1	24.6	100	100	112	100	79	79
AC-3 min-2	5.3	3.9	4.6	23.9	10.3	13.3	100	38	67	100	36	54
AC-3 min-3	4.0	3.2	3.7	22.2	13.4	16.0	100	45	110	100	27	103
AC-3 min-Aver.	4.3	3.7	4.2	25.4	16.2	18.0	100	61	96	100	47	78
AC-15 min-1	3.2	1.5	2.0	27.1	12.5	11.1	100	25	38	100	18	25
AC-15 min-2	6.6	3.7	5.1	25.2	16.5	19.4	100	45	76	100	24	53
AC-15 min-3	2.6	1.2	2.1	21.4	15.4	20.0	100	72	86	100	86	94
AC-15 min-4	5.0	3.2	3.0	24.6	14.0	19.8	100	59	59	100	60	85
AC-15 min-Aver.	4.4	2.4	3.1	24.6	16.6	17.6	100	50	64	100	50	64

Calculations of integrated areas are available from the authors upon request.

occurring during the 15-minute additional perfusion. The results of Experiment KC-15-2 are plotted in Fig. 2 as an example of this severe myocardial depression.

TABLE II. POTASSIUM CITRATE CARDIOPLEGIA

EXPERIMENT NUMBER	MAXIMUM STROKE WORK (GM.-METERS)						PER CENT FUNCTION					
	RIGHT			LEFT			RIGHT			LEFT		
	I	II	III	I	II	III	I	II	III	I	II	III
KC-3 min-1	4.4	3.7	3.2	31.2	24.2	21.3	100	83	74	100	74	57
KC-3 min-2	4.1	5.1	4.9	40.0	28.6	28.5	100	144	83	100	75	54
KC-3 min-3	4.1	3.7	3.1	20.5	19.8	19.3	100	97	90	100	76	92
KC-3 min-Aver.	4.2	4.1	3.7	30.6	20.9	23.0	100	108	82	100	75	68
KC-15 min-1	3.4	4.0	2.9	22.6	21.4	28.2	100	86	80	100	54	127
KC-15 min-2	4.5	1.6	2.0	26.8	4.5	7.7	100	38	47	100	16	19
KC-15 min-3	5.1	4.7	4.5	33.3	28.2	28.4	100	90	97	100	86	90
KC-15 min-4	3.3	2.0	2.9	26.4	15.8	18.7	100	54	88	100	52	68
KC-15 min-Aver.	4.1	3.1	3.1	27.2	17.8	20.8	100	67	78	100	52	76

Hypoxic arrest was achieved by simple aortic occlusion in 4 normothermic dogs. Cardiac action ceased on an average of 6 minutes after occlusion. After 15 minutes of occlusion, the aorta was released and cardiac action resumed spontaneously in 3 hearts. In the fourth heart, electrical defibrillation was easily accomplished. Table III shows conversion of post-arrest function curves

TABLE III. HYPOXIA (AORTIC OCCLUSION)

EXPERIMENT NUMBER	MAXIMUM STROKE WORK (GM.-METERS)						PER CENT FUNCTION					
	RIGHT			LEFT			RIGHT			LEFT		
	I	II	III	I	II	III	I	II	III	I	II	III
X-15 min-1*	3.6	0.9	0.9	38.0	10.7	6.7	100	29	25	100	28	20
X-15 min-2	5.0	2.4	3.0	24.2	19.0	17.5	100	52	60	100	67	68
X-15 min-3	5.6	3.1	3.4	29.1	24.4	31.1	100	50	54	100	69	104
X-15 min-4	8.6	3.8	5.6	26.9	10.3	17.2	100	48	66	100	32	34
X-15 Aver.	5.7	2.6	3.2	29.6	16.1	18.6	100	45	51	100	49	55

*Electrical defibrillation.

to per cent of control function. Ventricular depression occurred in the same range as following 15-minute arrest by acetylcholine and potassium citrate. It should be noted, however, that the hypoxic arrested hearts were quiet for an average of only 9 minutes, and recovery from hypoxic arrest is more delayed than that following arrest by the drugs tested. Experiment X-15-2 is plotted in Fig. 3 as an example of myocardial depression from hypoxic arrest.

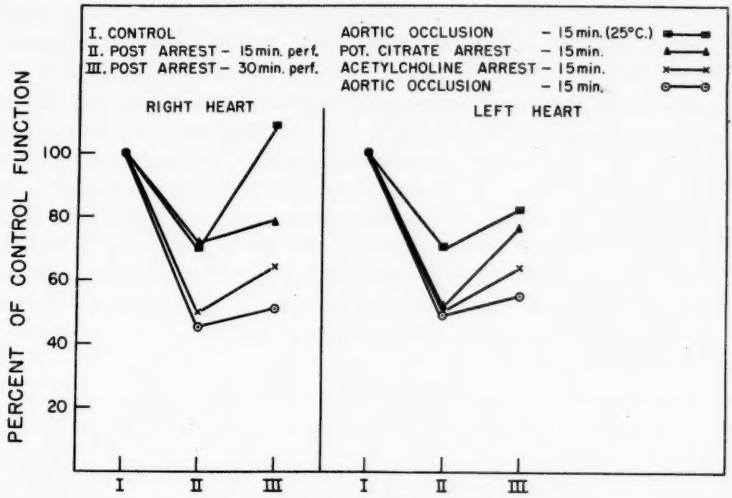

Fig. 7.—Ventricular function following cardioplegia.

Aortic occlusion at 25° C. for 15 minutes (during which time the animal was rewarmed and the heart remained below 29° C.) was performed in 4 experiments. As in the other experiments, myocardial function was measured at 35° C. In each case the heart moved quietly until release of the aorta, when good cardiac action resumed immediately. Post-arrest function curves were converted to per cent of control of function (Table IV). In this series of experiments, both ventricles showed less depression and more rapid recovery than was found in any of the three previously tested methods of cardioplegia. Fig. 4 illustrates the favorable influence of hypothermia as determined in Experiment 25-15-2.

TABLE IV. HYPOXIA (AORTIC OCCLUSION AT 25° C.)

EXPERIMENT NUMBER	MAXIMUM STROKE WORK (GM.-METERS)						PER CENT FUNCTION					
	RIGHT			LEFT			RIGHT			LEFT		
	I	II	III	I	II	III	I	II	III	I	II	III
25-15-1	10.4	6.1	7.5	42.3	26.6	23.6	100	82	87	100	83	68
25-15-2	7.5	5.1	6.1	40.5	23.0	22.2	100	87	115	100	52	56
25-15-3	11.1	8.1	6.7	33.1	22.3	28.4	100	68	69	100	76	114
25-15-4	2.9	1.9	3.6	45.7	30.4	31.3	100	43	158	100	69	86
25-15-Aver.	7.8	5.3	6.0	40.4	25.6	21.4	100	70	108	100	70	82

Results of the four techniques considered thus far are summarized in Fig. 7. Approximately 50 per cent loss of function occurs in both ventricles after 15-minute cardioplegia by each technique except for aortic occlusion at 25° C., where 70 per cent of function is retained by both ventricles. After an additional 15-minute perfusion, further recovery occurs in all cases: to 51 per cent and 55 per cent in the right and left ventricles, respectively, after simple aortic occlusion, 64 per cent and 64 per cent after acetylcholine, 78 per cent and 76 per cent after potassium citrate, and 108 per cent and 82 per cent after hypthermic aortic occlusion. The last method appears to be the least harmful.

Sympathetic stimulation: In 12 dogs, a fourth function curve was derived immediately after obtaining Curve III (on an average of 80 minutes post-arrest). During this phase of the experiment, the isolated right stellate ganglion was stimulated continuously with 20 to 30 volts at 25 cycles per second. The purpose of this maneuver was to determine whether elective cardiac arrest impaired the heart's responsiveness to sympathetic stimulation. In 75 per cent of cases (see Fig. 3), Curve IV revealed improvement over Curve III but, because control stimulation curves were not made, it is not known whether the additional improvement was spontaneous or induced. More conclusive studies are now in progress.

Total body hypothermia to 15° C. with interruption of perfusion for 15 minutes was performed in 3 dogs. Cooling and rewarming was effected by a heat exchanger in the extracorporeal circuit. In each experiment a quiet but tonic heart was produced, and in only 1 case was defibrillation necessary upon recovery. The results shown in Table V reveal wide discrepancies with a general tendency toward delayed loss of function. These findings may represent the delayed effect of hypothermia and rewarming. Since no metabolic studies were performed in this series of experiments, the data allow no conclusions.

TABLE V. HYPOTHERMIA (15°)

EXPERIMENT NUMBER	MAXIMUM STROKE WORK (GM.-METERS)						PER CENT FUNCTION					
	RIGHT			LEFT			RIGHT			LEFT		
	I	II	III	I	II	III	I	II	III	I	II	III
15-15-1	3.6	2.9	2.8	24.7	18.5	21.2	100	64	46	100	55	64
15-15-2*	2.8	4.2	4.3	18.9	15.5	16.5	100	142	139	100	101	102
15-15-3	9.8	2.7	7.8	23.3	14.2	12.0	100	59	52	100	56	28
15-15-Aver.	5.4	3.3	4.9	22.3	16.1	16.6	100	88.3	79.0	100	70.7	64.7

*Electrical defibrillation.

DISCUSSION

Investigations on cardioplegic agents have been carried out by a variety of methods. These include observations on the recovery and contractility of the isolated heart or on the isolated heart metabolically supported by a donor animal. Studies have been reported on the period of time for pump support following cardiac arrest in order to achieve a satisfactory and stabile arterial

pressure. Other investigations concern the oxygen consumption of the arrested heart or the developing tissue acidosis. Survival statistics have been compared between animals undergoing cardiac arrest and those in which coronary perfusion was uninterrupted, or interrupted by simple aortic occlusion. Finally, histologic changes in the myocardium of surviving animals have been reported. As compared to these methods, ventricular function studies by the Sarnoff technique have the distinct advantage of stressing the heart to the point of maximum performance while allowing a rough quantitation of this performance. Moderate or even minor impairment of myocardial function is thus revealed.

In a series of recent reports, Hanlon and his associates[5, 6] have shown marked depression of left ventricular myocardial function after a 30-minute arrest with either potassium citrate, acetylcholine, a mixture of prostigmine, magnesium sulfate and potassium citrate, or aortic occlusion. There was reduction in maximum stroke work performance from the control value of 25 to 50 to less than 10 Gm.-meters of work, when measured 10 minutes or 2 hours post-arrest. Less depression was noted after potassium arrest for 20 minutes. Partial protection of the heart was provided by previous digitalization or cooling to 28° C., and partial recovery was effected by administration of Wyamine. Weirich, Jones, and Burke[7] have noted a similar effect on myocardial function with increasing deterioration as the period of arrest was prolonged from 5 to 20 minutes. The effect of potassium citrate, acetylcholine, or simple aortic occlusion was roughly the same.

Braunwald, Morrow, and their associates[8] found severe depression in myocardial function after 20 to 30 minutes of arrest with either potassium citrate or acetylcholine. Using intermittent aortic occlusion, however, with 4 minutes of occlusion and 1 minute of release alternately over a period of 30 minutes, they found no significant depression.

The ventricular function studies reported in this paper were commenced before the work of the above investigators was known to us. Our methods and our results are closely similar to those of these authors. However, a valid method of integrating the data of identical experiments has been devised, permitting us to compare statistically the results of any given type of arrest in a series of experiments (Fig. 5). The data obtained were thus analyzed and justify the following conclusions.

CONCLUSIONS

1. Cardiac arrest for 15 minutes by any of the techniques studied results in depression of myocardial function to 50 to 70 per cent of control function when measured 15 minutes post-arrest.

2. Partial recovery of myocardial function occurs with an additional 15-minute perfusion.

3. At normal body temperature, 15-minute cardiac arrest by potassium citrate and by acetylcholine, and 15-minute aortic occlusion, cause similar degrees of depression, although recovery appears to be most rapid after potassium citrate and most delayed after hypoxia.

4. Prolongation of cardioplegia results in greater depression of myocardial function, and further delay in recovery of function.

5. Hypothermia to 25° C. partially protects the heart rendered hypoxic for 15 minutes and, in comparison with other methods studied, cardioplegia by this technique resulted in least depression and best recovery of myocardial function.

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REFERENCES

1. Melrose, D. G., Dreyer B., Bentall, H. H., and Baker, J. B. E.: Elective Cardiac Arrest, Lancet 2: 21, 1955.
2. Baker, J. B. E., Bentall, H. H., Dreyer, B., and Melrose, D. G.: Arrest of Isolated Heart With Potassium Citrate, Lancet 2: 555, 1957.
3. Sarnoff, S. J., and Berglund, E.: Ventricular Function. I. Stirling's Law of the Heart Studied by Means of Simultaneous Right and Left Ventricular Function Curves in the Dog, Circulation 9: 706, 1954.
4. Stirling, G. R., Stanley, P. H., and Lillehei, C. W.: The Effects of Cardiac Bypass and Ventriculotomy Upon Right Ventricular Function, S. Forum 8: 433, 1957.
5. Cooper, T., Willman, V. L., Zafiracopoulos, P., and Hanlon, C. R.: Myocardial Function After Elective Cardiac Arrest During Hypothermia, Surg. Gynec. & Obst. 109: 423, 1959.
6. Willman, V. L., Cooper, T., Zafiracopoulos, P., and Hanlon, C. R.: Depression of Ventricular Function Following Elective Cardiac Arrest With Potassium Citrate, Surgery 46: 792, 1959.
7. Weirich, W. L., Jones, R. W., and Burke, M.: Effect of Induced Cardiac Arrest by Potassium Citrate and Acetylcholine on Ventricular Function. Presented at Clinical Congress, American College of Surgeons, Atlantic City, N. J., Sept. 29, 1959.
8. Braunwald, N. S., Waldhausen, J. A., Cornell, W. P., Bloodwell, R. D., and Morrow, A. G.: Left Ventricular Function Following Elective Cardiac Arrest. Presented at Scientific Session of the American Heart Association, Philadelphia, Pa., Oct. 23, 1959.

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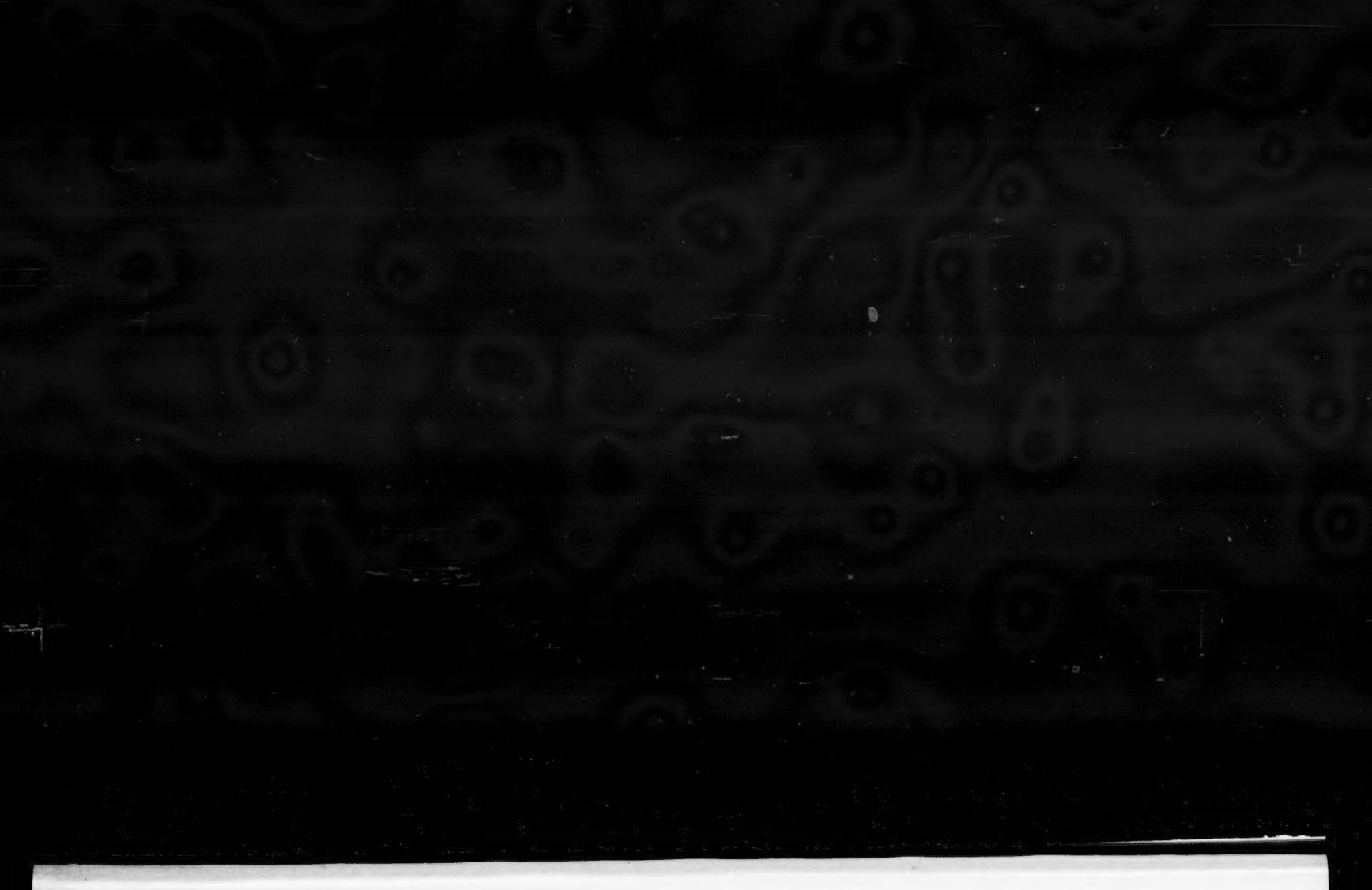
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